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of the
American Veterinary Medical Association
Formerly AMERICAN VETERINARY REVIEW
(Original Official Organ U. S. Vet. Med. Assn.)
H. Preston Hoskins, Secretary-Editor, 1230 W. Washington Blvd., Chicago, Ill.

R. R. DYKSTRA, President, Manhattan, Kan. M. JACOB, Treas., Knoxville, Tenn.

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Vol. LXXXI, N. S. Vol. 34 **AUGUST, 1932** **No. 2**

AGENDA

An outline of the program of the Atlanta convention appears on pages following, together with a list of the papers to be presented before the several sections. There are just fifty of these papers listed to date, with the possibility of a few more to be added at the last minute, which indicates the splendid efforts put forth by our section officers to secure program material for the meeting. It might indicate also that the economic depression had not had any deterring influence on veterinary investigational work and scientific literary production. The newly-created Section on Military Medicine will function for the first time. The Section on Poultry will hold only one session alone, meeting with the Section on Research for a joint session on Thursday.

At the Thursday evening session, Dr. C. W. Warburton, Director of Extension Work, U. S. Department of Agriculture, will deliver an address on "Extension Work and the Veterinarian." Dr. J. E. Shillinger, Bureau of Biological Survey, U. S. Department of Agriculture, will give an illustrated talk on "Disease Aspects of Wild Life Administration."

Among the important business items will be the report of the special committee that has been working on plans for affiliating state and provincial associations with the A. V. M. A. The most recent report available is to the effect that about twenty state associations have approved the tentative plans for affiliation.

Several of the standing committees have had a very busy year and interesting reports may be expected covering these activities. There may be quite a fight for the 1933 convention, as several cities have shown a very strong desire for the next meeting. The political pot has been boiling for some time and this fact will undoubtedly increase the attendance and the interest in the meeting. Elaborate plans have been made for the clinic to be held on Friday.

On to Atlanta.

BERNHARD BANG

Professor Bernhard Bang, of Copenhagen, Denmark, an honorary member of the A. V. M. A., died June 22, 1932, aged 84 years. Those who attended the Philadelphia convention in 1927 had the pleasure of meeting Professor Bang and hearing him deliver two addresses on that occasion. He is best known for his work in connection with tuberculosis eradication and his discovery of *Bacillus abortus* as the etiological factor in the widespread infection generally known as Bang's disease.

In 1928 a list of the literary productions of Professor Bang, consisting of 129 titles, was compiled and published in a volume entitled "Festskrift til Bernhard Bang," the occasion being the 80th birthday of the renowned scientist. The work contained eleven scientific articles by prominent investigators in the field of veterinary medicine.

IS THE VETERINARY CURRICULUM BECOMING HARDER?

Of the 933 freshman veterinary students registered for the five years 1923-4 to 1927-8, inclusive, approximately 83 per cent were graduated at the end of four years (classes of 1927 to 1931, inclusive). In the fall of 1928 there were 324 freshmen registered in the thirteen veterinary colleges in the United States and Canada. This represented an increase of about one-third over 1927, the greatest increase for some time. Of these 324 matriculants, only 247 were graduated this year. The percentage is 75.6, the lowest in six years. Is the veterinary curriculum becoming more strenuous, or, are our veterinary colleges becoming more exacting?

Based on this year's showing, only three out of four veterinary students can expect to be graduated on time. If the same rate of mortality holds next year, we may expect about 263 graduates for the year 1933. As a matter of fact, the number will probably be less, as there were only 270 junior students enrolled the past

year. However, this number may be augmented by a few who failed to finish on schedule this year. In any event we are still several years away from the time when the number of graduates will equal the theoretical requirements for the country.

NORTH DAKOTA PLACED ON HONOR ROLL

North Dakota was designated a modified accredited area on July 1, and thereby became the eighth state in the Union, and incidentally the second west of the Mississippi River (Idaho was the first), to be tuberculosis-free. The work of systematically eradicating bovine tuberculosis in North Dakota was started in 1918. Three counties (Foster, Eddy and Wells) were cleaned up in 1924 and at that time became tuberculosis-free areas. On July 1 of this year the last four counties (Billings, Logan, McIntosh and Sioux) reached the coveted goal and thereby enabled the entire state to become a tuberculosis-free area. Approximately 2,900,000 tuberculin tests were applied in the process of accreditation. Throughout the entire period two prominent veterinarians directed the work, Dr. W. F. Crewe, State Veterinarian, and Dr. H. H. Cohenour, U. S. Bureau of Animal Industry, inspector-in-charge.

The honor roll now reads:

North Carolina	Wisconsin
Maine	Ohio
Michigan	Idaho
Indiana	North Dakota

ATLANTA HOTELS

Hotel	Rooms	Rates	
		Single with Bath	Double with Bath
Atlanta-Biltmore (Official headquarters)	600	$3.00 Up	$5.00 Up
Ansley	400	$2.50 Up	$3.50 Up
Cox-Carlton	150	$2.50 Up	$4.00 Up
Georgian Terrace	300	$2.50 Up	$5.00 Up
Henry Grady	500	$2.50 Up	$4.50 Up
Piedmont	450	$2.50 Up	$4.50 Up
Winecoff	200	$2.00 Up	$4.00 Up

Other announcements in connection with the convention will be found on page 208.

Be sure and have your 1932 A. V. M. A. membership card with you at Atlanta.

OUTLINE OF PROGRAM

MONDAY, AUGUST 22, 1932

MORNING . . . . Committee meetings, 10 a. m.

AFTERNOON . . Meeting of Executive Board, 2 p. m.

EVENING . . . . Meeting of Southern States Veterinary Medical Association, 8 p. m. *Georgian Ballroom.*

TUESDAY, AUGUST 23, 1932

MORNING . . . . Registration. Opening session, 10 a. m., *Georgian Ballroom.* Address of Welcome. Response. President's address. Ladies attend.

AFTERNOON . . General session, 2 p. m., *Georgian Ballroom.* Reports of Executive Board, committees and officers. Nominations of officers. Ladies attend theatre party.

EVENING . . . . Meeting of college alumni groups, 6 p. m. President's reception and dance, 9 p. m.

WEDNESDAY, AUGUST 24, 1932

MORNING . . . . Sections meet, 9 a. m. Papers. Election, 9 a. m. to 6 p. m. Women's Auxiliary breakfast, 9 a. m.

AFTERNOON . . General session, 2 p. m., *Georgian Ballroom.* Reports of Executive Board and committees. Ladies take sight-seeing trip to Stone Mountain.

EVENING . . . . Banquet, 7 p. m., followed by dance.

THURSDAY, AUGUST 25, 1932

MORNING . . . . Sections meet, 9 a. m. Papers. Sight-seeing trip (North Side) for ladies, followed by luncheon, Druid Hills.

AFTERNOON . . General session, 2 p. m., *Georgian Ballroom.* Reports of Executive Board and committees. New business. Meeting of Southern Railway Veterinarians. Ladies attend bridge party, Druid Hills. Golf and swimming.

EVENING . . . . General session, *Georgian Ballroom.* Papers. Installation of officers. Entertainment.

FRIDAY, AUGUST 26, 1932

MORNING . . . . Clinic, 9 a. m., Fair Grounds. Ladies attend cycloramic view of Battle of Atlanta, Grant Park. Barbecue luncheon, 11:30 a. m., Fair Grounds. All attend.

AFTERNOON . . Clinic continued, 1 p. m., followed by entertainment.

EVENING . . . . Open.

PAPERS

SECTION ON GENERAL PRACTICE

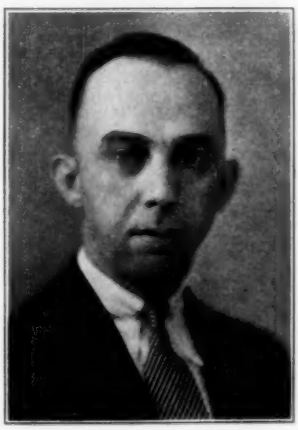
First Session

Wednesday Morning, August 24, 9 A. M.

Georgian Ballroom

1. The Value of Systemic Alkalinization in Influenzal Types of Diseases—Dr. R. W. Hixson, Falls City, Neb.
2. Lameness and Its Treatment in the Horse—Dr. John Baird, Goldstream Stud, Lexington, Ky.
3. Udder Diseases in Cattle—Dr. D. H. Udall, N. Y. State Veterinary College, Ithaca, N. Y.
4. Salt Sick: Its Cure and Prevention in Cattle—Dr. A. L. Shealy, University of Florida, Gainesville, Fla.
5. The Correlation Between Certain Properties of the Milk and the Type of Inflammation in Acute Mastitis—Drs. R. B. Little and F. S. Jones, Rockefeller Institute for Medical Research, Princeton, N. J.

DR. M. R. BLACKSTOCK
Chairman, Section on General Practice



DR. J. LEE HOPPING
Secretary, Section on General Practice

Second Session

Thursday Morning, August 25, 9 A. M.

Georgian Ballroom

6. Parasites in the Horse—Dr. W. H. Wright, U. S. Bureau of Animal Industry, Washington, D. C.
7. General Practice—Dr. J. M. Sutton, State Veterinarian, Atlanta, Ga.

8. Sterility in Cattle—Dr. W. A. Barnette, Greenwood, S. C.
9. Idiopathic Hemorrhagic Hepatitis of Swine—Drs. A. H. Quin, Jr., Des Moines, Iowa, and J. D. Shoeman, Dallas Center, Iowa.
10. Pasture Diseases in Sheep—Dr. W. H. Lytle, State Veterinarian, Salem, Ore.

DR. W. H. LYTLE
Chairman, Section on Sanitary Science and Food Hygiene

DR. J. V. KNAPP
Secretary, Section on Sanitary Science and Food Hygiene

SECTION ON SANITARY SCIENCE AND FOOD HYGIENE

First Session

Wednesday Morning, August 24, 9 A. M.

Room 6 (Mezzanine floor)

11. Establishing Accredited Abortion-Free Herds—Dr. C. A. Cary, State Veterinarian, Auburn, Ala.
12. The Relation of the Veterinary Profession to Municipal Meat Inspection—Dr. E. D. King, Jr., Board of Health, Valdosta, Ga.
13. Municipal Dairy Inspection—Dr. W. D. Martin, Albany, Ga.
14. Edward Jenner, the Father of Vaccination—Dr. C. N. McBryde, Bureau of Animal Industry, U. S. Department of Agriculture, Ames, Iowa.

Second Session

Thursday Morning, August 25, 9 A. M.

Room 6 (Mezzanine floor)

15. Handling of Anthrax—Dr. C. A. Cary, Albama Polytechnic Institute, Auburn, Ala.

16. Blackleg Aggressin Immunization—Drs. John Reichel and J. E. Schneider, Mulford Biological Laboratories, Sharp and Dohme, Glenolden, Pa.
17. Swine Sanitation—Dr. H. B. Raffensperger, U. S. Bureau of Animal Industry, Moultrie, Ga.

DR. F. W. MORGAN
Chairman, Section on Small Animals

DR. C. F. SCHLOTTHAUER
Secretary, Section on Small Animals

SECTION ON SMALL ANIMALS

First Session

Wednesday Morning, August 24, 9 A. M.

Silver Room

18. Critical Tests of the Efficacy of Single Treatments with Tracheal Brushes in the Mechanical Removal of Lung Worms from Foxes—Drs. K. B. Hanson and Frank G. Ashbrook, Bureau of Biological Survey, U. S. Department of Agriculture, Washington, D. C.
19. Intravenous Medication—Dr. E. J. Frick, Kansas State College, Manhattan, Kan.
20. Black Tongue—Dr. H. Calvin Rea, Charlotte, N. C.
21. Ethical Commercialism—Dr. I. M. Hays, Alabama Polytechnic Institute, Auburn, Ala.

Second Session

Thursday Morning, August 25, 9 A. M.

Silver Room

22. The Rabbit and Cavy Industry—Dr. H. Wood Ayers, Oklahoma City, Okla.

23. The Hospitalization and Care of the Cat—Dr. H. W. Brown, Fort Wayne, Ind.
24. Some Cases of Diabetes in Dogs—Dr. H. J. Milks, N. Y. State Veterinary College, Ithaca, N. Y.
25. Pre- and Post-Operative Care of Small Animals—Dr. C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

DR. W. A. HAGAN
Chairman, Section on Research

DR. C. C. PALMER
Secretary, Section on Research

SECTION ON RESEARCH

First Session

Wednesday Morning, August 24, 9 A. M.

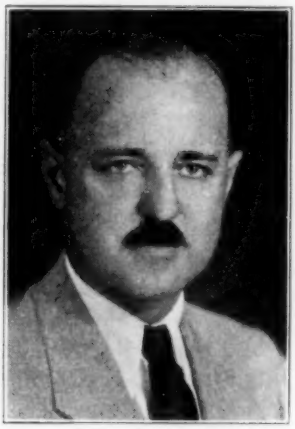
Room 10 (Mezzanine floor)

26. Further Studies in Bovine Coccidiosis—Dr. I. D. Wilson, Virginia Polytechnic Institute, Blacksburg, Va.
27. Studies on Canine Distemper. III. A Comparison of Natural and Experimental Virus Infections—Dr. A. S. Schlingman, Parke, Davis & Company, Detroit, Mich.
28. Some Observations on the Pathology of Johne's Disease (Illustrated)—Dr. E. T. Hallman, Michigan State College, East Lansing, Mich.
29. Further Studies on Brucella Infection in Swine—Drs. H. W. Johnson and I. F. Huddleson, Michigan State Experiment Station, East Lansing, Mich.
30. Studies on Brucella Infection at Meat Packing Plants. I. Incidence of Infection in Swine Slaughtered at Packing Plants. II. Incidence of Infection and Exposure in Workers at Packing Plants. III. The Effects of Meat

Preservation Processes on the Viability of *Br. Suis* in Infected Hog Meat—Drs. H. W. Johnson, I. F. Huddleson and E. E. Hamann, Michigan State Experiment Station, East Lansing, Mich.

31. A Continuation of the Study of the Etiology of Infectious Diarrhea (Winter Scours) in Cattle—Drs. F. S. Jones and Ralph B. Little, Rockefeller Institute for Medical Research, Princeton, N. J.
32. Relation of the Alcohol-Milk Test to Pregnancy in Cattle—Dr. C. C. Palmer, University of Delaware, Newark, Del.

DR. FRANK THORP, JR.
Chairman, Section on Poultry



DR. M. W. EMMEL
Secretary, Section on Poultry

SECTION ON POULTRY

First Session

Wednesday Morning, August 24, 9 A. M.

Room 14 (Mezzanine floor)

33. A Preliminary Report on the Study of Poultry Vermifuges—Dr. E. F. Thomas, University of Florida, Gainesville, Fla.
34. So-Called Range Paralysis—Dr. F. D. Patterson, Iowa State College, Ames, Iowa.
35. Some of the Practical Problems of a Poultry Practitioner—Dr. H. J. Seaman, Wauseon, Ohio.
36. Leukemia, Lymphatic Hyperplasia and Tumors Associated with Fowl Paralysis—Dr. E. P. Johnson, Virginia Polytechnic Institute, Blacksburg, Va.

37. The Use of Pigeon-Pox Virus as a Cutaneous Vaccine Against Fowl-Pox in an Egg-Laying Contest—Dr. M. W. Emmel and F. H. Orr, Jr., Alabama Polytechnic Institute, Auburn, Ala.
38. Disinfection of Avian Fecal Material—W. L. Mallmann and W. L. Chandler, Michigan State College, East Lansing, Mich.

SECTION ON RESEARCH AND SECTION ON POULTRY

Joint Session

Thursday Morning, August 25, 9 A. M.

Room 10 (Mezzanine floor)

39. A Liver Function Test in Sheep—Dr. J. N. Shaw, Oregon State Agricultural College, Corvallis, Ore.
40. A Discussion of Some Fundamental Principles and Practices Underlying the Application of the Agglutination Test for Bang's Disease (Illustrated)—Drs. C. P. Fitch and C. R. Donham, University of Minnesota, Saint Paul, Minn.
41. Treatment for Mastitis with Ultra-Violet Light, Formalin, Colloidal Carbon and Autogenous Bacterin—Dr. E. M. Gildow, H. C. Hansen and V. A. Cherrington, University of Idaho, Moscow, Idaho.
42. Infections of Fetuses and Foals—Dr. W. W. Dimock, University of Kentucky, Lexington, Ky.
43. Experiments on Fowl-Pox—Dr. C. L. Martin, University of New Hampshire, Durham, N. H.
44. Further Studies on the Efficiency of Vermifuges in Poultry—Dr. W. L. Bleecker and R. M. Smith, University of Arkansas, Fayetteville, Ark.
45. The Pathogenicity of the "Saprophytic Acid-Fast Bacteria"—Drs. W. A. Hagan and Philip Levine, N. Y. State Veterinary College, Ithaca, N. Y.

SECTION ON MILITARY MEDICINE

Thursday Morning, August 25, 9 A. M.

Room 14 (Mezzanine floor)

46. The Practicing Veterinarian and His Relationship to the Army—Lt. Col. D. S. Tamblyn, Chief, Canadian Army Veterinary Service, Kingston, Ont.
47. How Can the Operation of the Army Veterinary Service Be Improved in Future Engagements in the Light of Experience of the World War?—Maj. R. A. Kelser, V. C., U. S. Army, Medical Center, Washington, D. C.

48. Reminiscences of the Veterinary Service of the A. E. F.—Dr. L. A. Merillat, Chicago, Ill.
49. Some Lessons of Peace—Dr. N. S. Mayo, Highland Park, Ill.
50. Training Policies for Veterinary Reserve Officers—Dr. J. G. Horning, Houston, Tex.

MAJ. B. C. BRIDGES
Chairman, Section on Military Medicine

© H. & E.
DR. D. M. CAMPBELL
Secretary, Section on Military Medicine

CLINICS AND DEMONSTRATIONS

Friday Morning, August 26, 9:30 A. M.

Lakewood Fair Grounds

Dr. J. L. Hopping, Atlanta, Ga., *Chairman,* Sub-committee on Clinics

General Clinic

Demonstrations

1. DeLaval Magnetic Combine Milking System. H. L. Lykes, DeLaval representative, and Dr. J. G. Hardenbergh, Walker-Gordon Laboratories, Plainsboro, N. J.
   (This will consist of a complete unit, showing cows in the process of milking, handling of the milk and the sterilization.)
2. The Federal Hay Inspection Service and Its Relation to the Veterinary Profession. A. D. Harlan, Atlanta, Ga.
   (Exhibit of different grades of hay and grain.)

Special Clinics

Section I—Cattle

Dr. C. C. Rife, *Chairman,* Atlanta, Ga.

Demonstrations

1. Teat Surgery. Dr. C. H. Case, Akron, Ohio.
2. Rumeno-laparotomy. Dr. T. H. Ferguson, Lake Geneva, Wis.
3. Physical Examination of Dairy Cow, including technic of field diagnosis of mastitis. Dr. D. H. Udall, N. Y. State Veterinary College, Ithaca, N. Y.
4. Passing Stomach-Tube in Cow. Dr. T. H. Ferguson, Lake Geneva, Wis.
5. Intradermal Tuberculin Injection. Dr. C. C. VonGremp, Atlanta, Ga.
6. Drawing Blood Sample (Cow). Dr. C. C. Rife, Atlanta, Ga.
7. Sterility in Cattle, Including Drawing Semen from Bull. Drs. W. A. Barnette, Greenwood, S. C., and C. H. Case, Akron, Ohio.
8. Treatment of Metritis with Aid of Dural Block. Dr. W. K. Magill, Chester, S. C.

Section II—Swine

Dr. B. E. Carlisle, *Chairman,* Camilla, Ga.

Demonstrations

1. Autopsies and Diagnoses of Parasitic Diseases. Dr. H. B. Raffensperger, U. S. Bureau of Animal Industry, Moultrie, Ga.
2. Operations for Hernia and Otitis Media. Dr. W. F. Guard, Ohio State University, Columbus, Ohio.
3. Castration Technic (Knife and Emasculotome). Dr. W. R. Krill, Ohio State University, Columbus, Ohio.
4. Cesarean Operation (Sow). Dr. Frank M. Wilson, Mechanicsville, Iowa.
5. Epidural Anesthesia. Dr. W. R. Krill, Ohio State University, Columbus, Ohio.
6. Use of Stomach-Tube in the Pig. Dr. Chris E. Juhl, Osage, Iowa.

Section III—Small Animals

Dr. J. L. Ruble, *Chairman,* Orlando, Fla.

Demonstrations

1. Blood Transfusion. Dr. Ashe Lockhart, Kansas City, Mo.

2. Cecectomy. Dr. J. V. Lacroix, Evanston, Ill.
3. Pinioning Operation in Birds. Dr. J. E. Severin, Atlanta, Ga.
4. Bulla-Ossa Operation. Dr. W. F. Guard, Ohio State University, Columbus, Ohio.
5. Enucleation of the Eye. Dr. D. A. Eastman, Moline, Ill.
6. Spinal Anesthesia. Dr. E. J. Frick, Kansas State College, Manhattan, Kan.

SECTION IV—Horses

Dr. B. C. McLean, *Chairman*, Aiken, S. C.

Demonstrations

1. Roaring Operation (Standing Position). Dr. J. N. Frost, N. Y. State Veterinary College, Ithaca, N. Y.
2. Intraspinal and Intravenous Anesthesia. Dr. R. R. Dykstra, Kansas State College, Manhattan, Kan.
3. Tail Operations (Setting and Straightening Methods). Dr. John Baird, Goldstream Stud, Lexington, Ky.
4. Restraint in the Horse. Dr. John Salter, Dawson, Ga.
5. Autopsy on the Horse, Demonstrating the Various Species of Parasites. Dr. W. H. Wright, U. S. Bureau of Animal Industry, Washington, D. C.
6. X-Ray Demonstrations in the Horse. Dr. R. L. Humphrey, Middleburg, Va.

SECTION V—Poultry

Dr. M. W. Emmel, *Chairman*, Auburn, Ala.

Demonstrations

1. Culling Demonstration. Its advantages to the poultry practitioner. Dr. H. J. Seaman, Wauseon, Ohio.
2. Diagnosis of Intestinal Parasites. Administration of Anthelmintics. Dr. E. F. Thomas, University of Florida, Gainesville, Fla.
3. The Tuberculin Test, Reactions and Demonstration of Tuberculous Lesions. Dr. F. D. Patterson, Iowa State College, Ames, Iowa.
4. Whole-Blood Rapid Agglutination Test for Pullorum Disease. Dr. M. W. Emmel, Alabama Polytechnic Institute, Auburn, Ala.

   (Other diagnoses and autopsies showing various lesions associated with infectious and parasitic diseases as available material and time will permit.)

APPLICATIONS FOR MEMBERSHIP

(See July, 1932, JOURNAL)

FIRST LISTING

ALMQUIST, ELVIN W. Box 212, R. 1, Beaverton, Ore.
B. S., D. V. M., State College of Washington, 1932
Vouchers: E. E. Wegner and C. H. Seagraves.

BENDIX, WILMER L. Box 100, Dumbarton, Va.
B. V. Sc., Ontario Veterinary College, 1928
Vouchers: George C. Faville and I. D. Wilson.

CAMPBELL, DANIEL L. 820 N. E. Third Ave., Fort Lauderdale, Fla.
D. V. M., Alabama Polytechnic Institute, 1917
Vouchers: J. G. Catlett and John Wells.

CRAIGE, A. HENRY 1317 S. 57th St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1932
Vouchers: M. A. Emmerson and G. A. Dick.

DECAMP, CLAYTON E. c/o Rare Chemicals, Inc., Gray Oaks Ave., Nepera Park, N. Y.
D. V. M., Cornell University, 1924
Vouchers: R. R. Birch and C. E. Hayden.

HUFF, JOSEPH NEAL 4950 York St., Denver, Colo.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: B. R. McCrory and George H. Glover.

KING, JOSEPH J. 1100 N. St., Sacramento, Calif.
D. V. M., San Francisco Veterinary College, 1913
Vouchers: M. J. O'Rourke and J. M. Arburua.

MILLS, HENRY LEE 339 Colchester Ave., Burlington, Vt.
D. V. S., Grand Rapids Veterinary College, 1911
Vouchers: L. H. Adams and G. N. Welch.

O'CONNELL, JOHN A. Room 1205, 11 Beacon St., Boston, Mass.
M. D. V., Harvard University, 1896
Vouchers: Edward Knobel and Edw. T. Ryan.

PERRY, LUCIUS D. 20 Ferris St., Saint Albans, Vt.
B. V. Sc., Ontario Veterinary College, 1924
Vouchers: L. H. Adams and G. N. Welch.

RUMNEY, WILFRED J. 612 King St. W., Hamilton, Ont., Can.
B. V. Sc., Ontario Veterinary College, 1925
Vouchers: C. D. McGilvray and R. A. McIntosh.

Applications Pending

SECOND LISTING

(See July, 1932, JOURNAL)

Barnes, Charles F., c/o Interstate Packing Co., Winona, Minn.
Bechtol, Lauren L., Okolona, Ohio.
Coon, Elwyn W., Forest Grove, Ore.
Davis, Douglas L., 254 Capitol Place, Atlanta, Ga.
Gadd, John D., 512 S. 42nd St., Philadelphia, Pa.
Grinstead, Emil E., 207 E. 3rd St., Cle Elum, Wash.
Heagerty, Aloysius K., 5211 Windsor Mill Road, Baltimore, Md.
Knapp, John H., Ohio State University, Columbus, Ohio.
Ladson, Thomas A., Olney, Md.
Lockwood, Charles H., 905 New Jersey Ave. N. W., Washington, D. C.
Moyer, Raymond S., 609 Quebec St., Allentown, Pa.
Norling-Christensen, Olof, 1904 W. North Ave., Chicago, Ill.
Rohrer, Arthur A., 1852 N. Fourth St., Columbus, Ohio.
Starr, G. H., Box 56, National Stock Yards, Ill.
Sternfels, Mark, 560 W. 180th St., New York, N. Y.
Wastrack, William R., Cedarburg, Wis.

The amount which should accompany an application filed this month is $7.08, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Connecticut Veterinary Medical Association. Bridgeport, Conn. August 3, 1932. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. August 3, 1932. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

Hudson Valley Veterinary Medical Society. West Point, N. Y. August 10, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Tulsa County Veterinary Association. Tulsa, Okla. August 11, 1932. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. August 16, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. August 17, 1932. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.

American Veterinary Medical Association. Atlanta Biltmore Hotel, Atlanta, Ga. August 23-26, 1932. Dr. H. Preston Hoskins, Secretary, 1230 W. Washington Blvd., Chicago, Ill.

National Veterinary Medical Association of Great Britain & Ireland. 50th Annual Congress. Folkestone, Eng. August 8-13, 1932. F. Knight, Secretary, 2, Verulam Bldg., Gray's Inn, London, W. C. 1, Eng.

Eastern Iowa Veterinary Association. Mechanicsville, Iowa. September 6, 1932. Dr. Paul V. Neuzil, Secretary, Blairstown, Iowa.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. September 7, 1932. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. September 13, 1932. Dr. E. E. Sweebe, 14th St. and Sheridan Road, North Chicago, Ill.

Meat Inspection for Oakland, California

Several months ago the Health Department of Oakland, Calif., employed four veterinarians as additions to the meat inspection force, which also requires the services of thirteen lay inspectors. The veterinarians were given temporary appointments, pending a civil service examination, which was held July 9, 1932. Several other applicants also took the examination.

INVESTIGATIONS ON THE VIRULENCE OF VESICULAR STOMATITIS VIRUS AND THE PROPERTIES OF IMMUNE SERA*

By KURT WAGENER, *Berlin, Germany*
Hygienic Institute, Veterinary College

In special investigations on the passive immunity test in small animals,[1] it was established as a difference from foot-and-mouth disease that it is possible to protect small animals completely by passive immunization against an experimental infection with vesicular stomatitis. In these investigations it was found also that the amount and duration of protection afforded by the serum depend on the potency of the serum and the amount administered, as well as on the virulence of the virus used for the inoculation.

After the manner of the serum standardizations in foot-and-mouth disease (Waldmann and Pape,[2] Waldmann and Trautwein[3]), in vesicular stomatitis also the potency, or titre, of immune serum can be determined by means of the serum test in guinea pigs.[1] In foot-and-mouth disease the smallest quantity of serum which still prevents the generalization after the simultaneous inoculation is regarded as the titre of the serum.[2, 3] In contrast to this, in vesicular stomatitis the titre of an immune serum is the smallest serum dose which still prevents the cutaneous infection on one metatarsal pad when the serum is administered 24 hours before the inoculation. Among those factors that influence the results of serum tests, the conditions of the animal body may be restricted by always using animals of the same size, age and weight, and keeping them under equal conditions. In the writer's investigations on vesicular stomatitis, guinea pigs weighing 500 grams and white rats weighing 200 grams proved to give the best and most uniform results in serum tests. Furthermore, the results are widely influenced by the potency of the serum used for the test. In the investigations referred to[1] the writer used convalescent serum, that is serum collected four weeks or longer after the inoculation, as well as immune, or hyperimmune, serum which had been prepared by several injections with living virus after recovery of the animal from the first infection. In these experiments in which the writer considered first only the clearing up of the general principles, no complete serum titrations were made and it had

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not been determined whether there exist differences between the convalescent and hyperimmune serum of guinea pigs. After these reported findings, the writer endeavored to investigate the manner of the serum action and also the influence of the virus in these serum tests.

Some experiences and observations in the course of infection in foot-and-mouth disease led the writer to the supposition that the virus has a changeable virulence during the course of infection. This has been established also in vesicular stomatitis by Olitsky.[4] The following investigations are attempts to prove this supposition. For this purpose the virus of vesicular stomatitis (Indiana strain) was transferred every 24th hour instead of the usual way of transferring the virus at intervals of 48 hours, beginning with the 713th transfer in guinea pigs. The passage of the virus from one guinea pig to another at intervals of 24 hours was always successful, even when there were no vesicles to be seen, or any other indication of infection, when the virus was collected. In this way the Indiana virus was transferred without interruption for 105 days. Then the passage was discontinued intentionally. In order to avoid confusion, the virus transferred at intervals of 24 hours was called Indiana virus A, in contrast to that Indiana virus which was transferred at the usual intervals. This latter virus, which was transferred within one week, twice at intervals of 48 hours and once after 72 hours, was called Indiana virus B. These two viruses had been tested in regard to their virulence several times during the investigation. The results of the serum tests in guinea pigs were regarded as a standard of the virulence of these viruses. The test was to determine the smallest dose of serum which still prevents an inoculation with either of the viruses. Four different serum tests have been made for establishing, or comparing, the virulence of the viruses.

Experiment I

The first experiment consisted of two series, in each of which four guinea pigs were treated subcutaneously with serum doses of 0.2, 0.5, 1.0 and 2.5 cc, 24 hours before the inoculation on one metatarsal pad. One of the series received a 24-hour virus of the 62nd transfer of the Indiana virus A; the other series was inoculated with the 48-hour virus of the 61st transfer of the Indiana virus A. The results of the test, as recorded in table I, show that the infection with the 48-hour virus A was prevented with each of the serum doses administered, while even the largest dose (2.5 cc) of the same serum was not sufficient to protect

against the 24-hour virus. The normal serum used as a control did not give any protection. These findings indicate that the 24-hour virus had a higher virulence than the 48-hour virus.

TABLE I—*Results of test to demonstrate protective power of convalescent serum against 24-hour and 48-hour viruses.*

DAY	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8
	Convalescent Serum (Indiana)							
1	0.2	0.5	1.0	2.5	0.2	0.5	1.0	2.5
2	Inoculated cutaneously with Indiana virus A 62 (24-hour)				Inoculated cutaneously with Indiana virus A 61 (48-hour)			
5	+	+	+	−	−	−	−	−
7				−	−	−	−	−
10				+	−	−	−	−
15					−	−	−	−
18					−	−	−	−
32	Reinoculated with Indiana virus B (48-hour)							
34	−	−	−	−	−	−	−	−

+ = primary lesions.
− = no visible lesions.

EXPERIMENT II

While in the first experiment the Indiana virus A from two following passages was used, in the second experiment the 24- and 48-hour virus A of the 68th passage was compared with the 24- and 48-hour virus Indiana B of the 741st passage. For this purpose both series of guinea pigs were subcutaneously treated 24 hours before the inoculation with the same convalescent serum. Results of this second experiment (table II) proved again the higher virulence of the 24-hour virus compared with the 48-hour virus; but there was only a slight difference. While in the guinea pig treated with 2.0 cc of serum the virus B was able to produce only a very small vesicle at the site of inoculation, the virus A developed normal vesicles under the same conditions. No generalization was noticed in either of the series. In the case of the 48-hour virus, there was a marked superiority of the virus A compared with virus B. A complete protection was established with 0.1 cc of serum against virus B, while virus A required ten times as much serum for the same effect. The second experiment proved again the higher virulence of the 24-hour virus in contrast with the 48-hour virus.

TABLE II—*Results of test to demonstrate protective power of convalescent serum against viruses of the 68th and 74st passages.*

DAY	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8	GP 9	GP 10	GP 11	GP 12	GP 13	GP 14	GP 15	GP 16	GP 17	GP 18
	Convalescent Serum (Indiana)																	
1	0.2	0.5	1.0	2.0	0.1	0.3	0.5	1.0	2.0	0.2	0.5	1.0	2.0	0.1	0.3	0.5	1.0	2.0
	Indiana Virus A 68									Indiana Virus B 741								
2	24-hour				48-hour					24-hour				48-hour				
5	+	+	+	+	+	+	+	−	−	+	+	+	+	−	−	−	−	−
8								−	−					−	−	−	−	−
12								−	−					−	−	−	−	−
15								−	−					−	−	−	−	−
18								−	−					−	−	−	−	−
25	Reinoculated with Indiana Virus B (48-hour)																	
27	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

TABLE III—*Results of test to demonstrate protective power of convalescent serum against 24-hour, 48-hour and 72-hour viruses.*

DAY	GUINEA PIGS																					
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1	Convalescent Serum (Indiana)																					
	1.5	2.5	4.0	0.5	1.0	2.0	0.05	0.2	0.5	1.0	1.5	3.0	0.03	0.05	0.1	0.3	0.5	0.01	0.03	0.06	0.1	0.3
2	Indiana Virus A 80										Indiana Virus B 746											
	24-hour			48-hour			72-hour				24-hour		48-hour					72-hour				
4	+	+	−	−	−	−	+	+	−	−	+	−	+	+	+	+	+	+	+	+	+	−
5	+	+	+	−	−	−			−	−		−										−
8				−	−	−			−	−		−										−
10				−	−	−			−	−		−										−
12				−	−	−			−	−		−										−
15									−	−		−										−
20									−	−		−										−
24	Reinoculated with Indiana Virus B (48-hour)																					
26	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+

Experiment III

In this series the virulence test was extended to include the 72-hour virus of both strains. The results are shown in table III. A continuous decrease of the virulence from the 24-hour, over the 48-hour, to the 72-hour virus, is to be seen. The difference between virus A and virus B is not very definite in this experiment, because the serum doses administered in this test proved to be insufficient. Nevertheless, the higher virulence of the 24-hour virus A in comparison with the 24-hour virus B is noticeable, but not in the cases of the 48- and 72-hour viruses.

Experiment IV

In experiment IV the similar conditions of the New Jersey virus were tested, using the 24- and 48-hour viruses of the usual virus transfer 680 because no different virus transfers were made with the New Jersey strain. As in experiment II, 2 series of guinea pigs were treated with the same doses of serum 24 hours before the usual inoculation on the metatarsal pad. Results recorded in table IV show that in the New Jersey strain also the virulence of the 24-hour virus is higher than the 48-hour virus.

Table IV—*Results of test to demonstrate protective power of convalescent serum against 24-hour and 48-hour viruses.*

Day	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8	GP 9	GP 10
1	Convalescent Serum (New Jersey)									
	0.2	0.5	1.0	2.0	3.0	0.2	0.5	1.0	2.0	3.0
2	New Jersey virus 680									
	24-hour					48-hour				
4	+	+	+	−	−	+	+	+	+	+
5				−	−					
6				−	−					
8				−	−					
10				−	−					
13				−	−					
17				−	−					
22	Reinoculated with New Jersey virus (48-hour)									
24	−	−	−	+	+	−	−	−	−	−

The results confirm that the virus of vesicular stomatitis changes its virulence during the course of infection, as supposed at the beginning of the experiments. It is noteworthy

that the virus apparently has its highest virulence in the early stages of the infection. It seems that the virus does not need a complete development of the vesicle for gaining its highest degree of virulence. The size of the vesicle does not indicate the degree of virulence of the virus contained in the vesicle. Development of vesicle and degree of virulence do not run parallel. Considering that in vesicular stomatitis the generalization of the infection, which is generally regarded as the summit of the disease, usually takes place between the 5th and 7th days after the inoculation, it is remarkable that the virulence has already passed its highest point at this time. The summit of the virulence is attained at a moment when there are no, or only slight, indications of the specific infection in the guinea pig.

After having shown the possibility of a virulence test in vesicular stomatitis by means of the passive immunity test in guinea pigs, further investigations were made to test the manner of the serum action. The bases of the following experiments were the experiences in foot-and-mouth disease research showing that the foot-and-mouth disease immune serum develops viricidal action also *in vitro* (Bedson, Maitland, and Burbury[4, 5]).

In Vitro Tests

For testing the vesicular stomatitis immune serum, *in vitro* tests were made, examining the action of the serum in various dilutions on an equal amount of virus. This plan of experiment is different from that recorded by Bedson, Maitland, and Burbury,[4, 5] who used to mix, in their viricidal experiments on foot-and-mouth disease serum *in vitro*, equal amounts of serum with increasing virus dilutions. In the present experiments, in which the writer considered first only the clearing up of the general principles, he used the above-mentioned, simple plan of experiment because, in the manner of Bedson, Maitland, and Burbury,[4, 5] it is necessary to determine the smallest amount of virus that will still infect before the virus is used (Minett[6]). To various serum dilutions were added equal quantities of virus suspensions, thus doubling the dilutions. The virus suspension was prepared by grinding in a mortar the covers of vesicles from two guinea pigs with 20 cc of salt solution. This suspension was filtered through gauze and cotton before being mixed with the virus-serum dilutions, in order to hold back gross particles. The mixtures of serum and virus were kept in tubes for from one to twenty-four hours at different temperatures before the cutaneous inoculation on the metatarsal pad of guinea pigs.

TABLE V—*Results of test to demonstrate comparative protective powers of convalescent guinea-pig serum and normal guinea-pig serum. (Serum and virus mixed in vitro and kept 24 hours before injection.)*

DAY	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8	GP 9	GP 10	GP 11	GP 12	GP 13	GP 14
1	Convalescent Serum (Indiana)								Normal Serum (Indiana)					
	1:10	1:20	1:80	1:160	1:320	1:1280	1:2560	1:5120	1:10	1:80	1:160	1:640	1:2560	Virus control
2	−	−	−	+	−	+	+	+	+	+	+	+	+	+
4	−	−	−		−									
6	−	−	−		−									
8	−	−	−		−									
10	−	−	−		−									
16	−	−	−		−									
20	−	−	−		−									
30	Reinoculated with Indiana Virus (24-hour)													
32	−	+	−	−	−	−	−	−	−	−	−	−	−	−

Experiment I

In experiment I, the action of a convalescent guinea-pig serum was compared with normal guinea-pig serum. Both of the sera were tested in various dilutions, as recorded in table V, on equal quantities of virus during 24 hours at ice-box temperature. The immune serum destroyed the virus in dilutions up to 1:320, except the dilution of 1:160, while the normal serum showed no effect on the virus. By the reinoculation with the homologous strain, 29 days after the previous inoculation, all guinea pigs which had developed visible lesions proved to be immune, as well as those guinea pigs inoculated with the dilutions 1:10, 1:80 and 1:320, which had shown no visible lesions after the first inoculation. Only the guinea pig inoculated with the dilution 1:20 came down after the reinoculation.

Experiment II

In the second experiment, different viruses were tested *in vitro* against guinea-pig convalescent serum. The dilutions of serum and virus were kept only two hours at room temperature before their virulence was tested on guinea pigs. Results indicate that the 24-hour virus A was killed by the serum dilutions up to 1:80, while the 48-hour virus A was still killed by the serum dilutions up to 1:2,560 (table VI). This proves a distinctly lower virulence of the 48-hour virus A compared with the 24-hour virus A. The 48-hour virus B, tested in the same experiment, proved to be less virulent than the 24-hour virus A but stronger than the 48-hour virus A. It was destroyed by the serum dilutions up to 1:320, but not by the dilution 1:640. This shows clearly that the difference of virulence as shown in the passive immunity test can be demonstrated also *in vitro*.

Experiment III

Experiment III was a control on the results gained in experiment II. The tested viruses were suspensions of covers from each of two guinea-pig vesicles ground in a mortar with 15 cc of salt solution. The serum dilutions, which were changed a little in this experiment, acted for one hour at room temperature on equal amounts of virus. Results are recorded in table VII.

In this attempt the viruses were destroyed by the same serum as used in experiment II, but in still higher dilutions. The 24-hour virus A also showed the highest virulence in this test, but the 48-hour virus B was only slightly inferior. The 48-hour virus A showed the lowest virulence. It was distinctly inferior to either of the other viruses. The reinoculations indicated that

TABLE VI—*Results of test to demonstrate protective power of guinea-pig convalescent serum. (Serum and virus mixed in vitro and kept two hours before injection.)*

DAY	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8	GP 9	GP 10	GP 11	GP 12	GP 13	GP 14	GP 15	GP 16
	Indiana virus A										Indiana virus B					
1	24-hour					48-hour										
	Convalescent serum (Indiana)															
	1:20	1:80	1:160	1:320	1:640	1:40	1:160	1:320	1:1280	1:2560	1:80	1:320	1:640	1:1280	1:2560	1:5720
3	−	−	−	+	−	−	−	−	−	−	−	−	+	−	+	+
5	−	−	+		−	−	−	−	−	−	−	−		−		
8	−	−			−	−	−	−	−	−	−	−		−		
10	−	−			−	−	−	−	−	−	−	−		−		
16	−	−			−	−	−	−	−	−	−	−		−		
25	Reinoculated with Indiana virus B (48-hour)															
27	−	−	−	−	−	+	+	+	+	+	−	−	−	−	−	−

TABLE VII—*Results of control test to demonstrate protective power of guinea-pig convalescent serum. (Serum and virus mixed in vitro and kept one hour before injection.)*

DAY	GP 1	GP 2	GP 3	GP 4	GP 5	GP 6	GP 7	GP 8	GP 9	GP 10	GP 11	GP 12	GP 13	GP 14	GP 15
	Indiana virus A 88					Indiana virus A 87					Indiana virus B 748				
1	24-hour					48-hour									
	Convalescent serum (Indiana)														
	1:50	1:100	1:200	1:400	1:800	1:100	1:400	1:800	1:1600	1:3200	1:100	1:200	1:800	1:1600	1:3200
3	−	−	−	−	+	−	−	−	+	−	−	−	+	−	+
5	−	−	−	−		−	−	−		−	−	−		+	
8	−	−	−	−		−	−	−		−	−	−			
12	−	−	−	−		−	−	−		−	−	−			
15	−	−	−	−		−	−	−		−	−	−			
18	Reinoculated with Indiana virus B (48-hour)														
20	+	+	+	+	−	+	−	+	−	+	+	+	−	−	−

all guinea pigs which had shown no lesions after the previous inoculation had not become immune, except one guinea pig which proved to be immune without having yielded lesions after the first inoculation.

General Discussion

The investigations described indicate the differences principally in the virulence of the vesicular stomatitis virus during the course of infection in guinea pigs. The results show, in confirmation of investigations reported in another place,[1] that the passive immunity test in guinea pigs, after the modified manner of testing foot-and-mouth disease serum, can be used for testing the virulence of vesicular stomatitis virus. In four virulence tests it was shown that there are distinct differences in the virulence during the course of infection in guinea pigs. The virus collected 24 hours after the cutaneous inoculation, when no, or only slight, development of vesicles is visible, is evidently superior to the virus collected at later moments. The vesicular stomatitis virus develops its highest concentration or virulence before the visible eruption of vesicles. The findings of Olitsky[7] were confirmed experimentally by these investigations. For experimental work in vesicular stomatitis, especially for comparative studies, it is necessary that in the future the differences in the virulences of the virus be considered.

Attempts to cultivate the virus in a state of a higher virulence by the continuous transmission from one guinea pig to another at intervals of 24 hours evidently do not prove that it is possible to keep the virus at its higher virulence. It seems that by this manner the original stronger virus loses gradually in virulence when continuously transferred over a longer period. In a single guinea pig the virulence of such transferred virus decreases more quickly than the virus that is transferred at intervals of 48 hours. The 48-hour virus A proved to be not, or only slightly, superior to the 48-hour virus B; but this question is to be tested in further investigations before a definite judgment can be given.

The *in vitro* tests confirm the results of the passive immunity tests on the virus and immune serum properties. They demonstrate that the immune serum in vesicular stomatitis has viricidal properties which are shown also outside the body, and kill the virus in relatively large amounts completely, so that it loses its infectious and antigenic powers. Sometimes the virus is deprived only of its infectious, but not its antigenic, power by the serum, as shown by the immunity of the test guinea pigs after the reinoculation.

SUMMARY

1. The guinea-pig convalescent serum in vesicular stomatitis has viricidal properties which can be demonstrated *in vivo* and *in vitro*.

2. The passive immunity test, as well as the test *in vitro*, can be used for a standardization of the vesicular stomatitis virus and immune serum.

3. The virus of vesicular stomatitis develops its highest virulence about 24 hours after the cutaneous inoculation of the guinea pig, but before vesicle development has passed its peak. The size of the vesicle is not a measure of virulence.

4. It does not seem that the virus, when continuously transferred at its highest virulence from one guinea pig to another at intervals of 24 hours, can be kept at its original higher virulence. The natural decrease of virulence in such a virus seems to develop more quickly than in the virus which is transferred at intervals of 48 hours.

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THE HISTOPATHOLOGY OF CUTANEOUS AND SUBCUTANEOUS NODULES OF CATTLE*

By RUSSELL A. RUNNELLS, *Ames, Iowa*
Department of Veterinary Pathology, Iowa State College

The movement to eradicate tuberculosis from cattle in the United States was inaugurated in 1917. Progress of the work has been so rapid that in many parts of the country the disease has almost disappeared. At the beginning of the campaign, the disease was so widespread, and the animals often so extensively infected, that it was not difficult to demonstrate visible lesions when tuberculin-reacting animals were slaughtered. With repetition of the tuberculin test annually, and sometimes semi-annually, there finally came a time when it was not always so easy to find visible lesions. This necessitated not only a more thorough search for tubercles, but also a search for them in parts of the body which were formerly not examined at all, or at least only casually. The skin was one of these parts. Reports on skin tuberculosis in cattle in the United States prior to 1921 are rare.

Since the importance of skin tuberculosis has been realized, the question arises as to how much dependence can be placed upon the gross and microscopic appearance of cutaneous and subcutaneous nodules in arriving at a diagnosis of this condition. The practical purpose of this investigation is to attempt to answer this question by making a histopathological study of such nodules found in cattle that have reacted to the tuberculin test. Another purpose is to study the nature and development of these nodules.

REVIEW OF LITERATURE

Traum[1, 2, 3] reported on the gross pathology and bacteriology of several cases of a subcutaneous nodular disease that resembles tuberculosis, but which he designates as lymphangitis due to an acid-fast organism. Eight of his cases are included in this study.

Day[4] reported on the skin lesions of 85 tuberculin-reacting cattle. In over 55 per cent of them he found tuberculous cutaneous nodules, principally on the extremities and teats. He does not mention that their histopathology was studied, so it is presumed that the diagnosis was based upon their gross appearance, which he describes minutely.

Wright[5] studied nine cases of tuberculosis cutis in cattle. He

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states that tuberculosis verrucosa is the form of the disease usually caused by the bovine strain of the organism. To him a differential diagnosis is not difficult. He mentions specifically only epithelioma as giving any difficulty in this respect.

Hastings, Beach and Weber[6] made a bacteriological study of cutaneous nodules from 23 so-called "no lesion" tuberculin reacting cows. Even though seven of the 23 revealed acid-fast bacilli upon microscopic examination, they believe there is little justification for classing these lesions as tuberculous.

Carpenter and Goldberg[7] made a bacteriological and histopathological investigation of 30 cases. Their study is referred to again in this paper.

Marsh[8] studied histologically cutaneous tubercles from the teats of 16 tuberculin-reacting cows. Sections from 15 of them showed the characteristic histopathology of tuberculosis. Smears from 10 of these tubercles revealed acid-fast bacilli. An important observation was that none of the lesions showed caseation.

Day[9] examined 200 skin lesions from tuberculin-reacting cattle. Sections from many of these, without exception, showed histopathological changes of tuberculosis.

Mitchell,[10] in connection with a report on the bacteriology of subcutaneous tuberculosis, described in gross the character of the lesions found.

Crawford[11] observed in more than 20 cases that the lesions were subcutaneous, attached to the under layer of the skin but not properly a part of the skin. In only two cases was there a connection between the lesion and the skin.

Hagan,[12] in connection with a study of subcutaneous lesions which sometimes induce tuberculin hypersensitiveness in cattle, states that in the light of our present knowledge it cannot be proved that the skin-lesion disease of cattle is or is not a form of tuberculosis.

Elder, Lee and Phelps[13] investigated the bacteriology of 32 skin and 2 teat lesions that had the gross appearance of tuberculosis. They found acid-fast organisms in 91 per cent of them. Acid-fasts from 21 per cent of these lesions produced tuberculosis in laboratory animals. No report of a histopathological study of these lesions was given.

Graham, Thorp and James[14] studied sections from the subcutaneous lesions of two cows. One repeatedly failed to react to the intradermic tuberculin test but reacted to an intravenous injection of avian tuberculin. The other reacted to both tests. A brief description of the microscopic appearance of the lesions indicates that they were typical tubercles.

Material and Methods

Specimens for this study were obtained from cattle in Michigan, Arizona, California, Quebec, Montana, New York and Iowa. All of them came from tuberculin-reacting animals. Fifty head of cattle furnished 56 lesions.

Practically all of these lesions were removed from the skin of the extremities. Some of them occurred singly, others in patches or in chains. The smallest single nodule measured 5 mm (3/8 inch) in diameter. The largest patch of nodules covered an area of 100 square centimeters (16 square inches). Most of them were wholly within the hide. A few were attached, but could be dissected easily from the under side of the hide. Their contents varied. A few contained semi-fluid, and several a heavy, light yellow pus with calcareous granules. Others had thick, fibrous capsules with septa-like, fibrous bands dividing them into smaller subdivisions, all of which were filled with inspissated, yellow, granular material. The smaller ones containing no pus resembled fibrous neoplasms. The epidermis over the nodules was usually intact and in most cases hair was present. In a few cases a scar was visible. Only one of the Michigan lesions discharged pus. Judging from the appearance of the Arizona lesions they did not. Traum reports that some of the California lesions did. The skin over the Quebec and New York cases was intact. This was evidently the case with the Montana specimens also. Hair was absent over one of the Iowa lesions.

Michigan specimens: All specimens of this series were obtained in abattoirs in Detroit, Michigan. The specimens were collected on days when tuberculous animals were being slaughtered. As soon as a hide was removed it was spread out upon the floor for examination. The nodules were detected mostly by palpation. Several of them were so small that they would easily escape a visual examination. Most of these lesions would be classed as those of tuberculosis if one relied entirely upon their gross appearance in making a diagnosis. In none of the cases was there any lymph-gland involvement in the region of the nodules.

Specimens from 18 animals were collected. These consisted of single nodules, chains of nodules, and in one case a patch of nodules. In all there were 22 lesions. The exact number of hides examined to obtain these lesions is not known. On one day, however, 11 specimens were obtained from 54 hides. Four animals from which lesions were obtained showed no other changes resembling tuberculosis.

Arizona specimens: These specimens were collected at Phoe-

nix, Arizona, and sent in borax to Blacksburg, Virginia, where they were examined. The principal object in obtaining lesions from Arizona was that it was thought that pseudotubercles due to cactus spines might be encountered. Four animals furnished six lesions.

California specimens: Dr. Jacob Traum, at the University of California, furnished these eight specimens, which had been used in his study of the bacteriology of the subcutaneous, nodular disease resembling tuberculosis, which he designates lymphangitis. He demonstrated acid-fast organisms in pus smears. With two exceptions, these specimens had been preserved in Kaiserling I, in some instances for several years.

Quebec, Montana and New York specimens: One specimen from Quebec, five from Montana, and three from New York were furnished by investigators[6,7,9] who have extensively studied various phases of cutaneous and subcutaneous tuberculosis of cattle. The Quebec specimen came in Kaiserling solution. It was the only one resembling tuberculosis found in the affected animal. Other animals in the herd were affected with tuberculosis. Those from Montana had been fixed in formalin and embedded in paraffin. Those from New York were sent in formalin. No history accompanied them.

Iowa specimens: These eleven lesions were removed from Iowa reactors that were slaughtered under federal supervision at Albert Lea, Minnesota. No other visible lesions were found. They were located on the limbs. The fresh specimens were sent to Ames, Iowa, where they were examined.

TECHNIC

The normal skin of the bovine is from 3 to 7 mm thick. When involved as it is in all the specimens in this study, it is much thicker and usually much more dense. Therefore, the fixation and hardening processes make the skin leather-like and render sectioning difficult. By fixing in formalin for a period not exceeding 24 hours and by clearing in cedarwood oil or chloroform, this difficulty was partly overcome. Several of the specimens had to be decalcified. Sections of all the lesions were stained with hematoxylin and eosin. Van Gieson's stain also was used on the California cases. Sections representing the three principal types of lesions encountered were stained according to the Starry method* for acid-fast microörganisms. Pus-smears from several cases also were stained for acid-fasts.

*A method used in the Pathological Laboratory at the University of Michigan.

HISTOPATHOLOGY

Inasmuch as the nodules studied may be grouped into three principal classes, a detailed description of each lesion necessitates considerable repetition because of the similarity of histological changes in each group. Therefore, only a typical nodule of each class will be described.

FIG. 1. Pyogenic abscess involving subcutis but encroaching upon the cutis. While the structure of the lesion at present is characteristic of that produced by a pyogenic infection, the single giant-cell may indicate a tuberculous origin.

Pyogenic abscess: Three lesions from the region of the metatarsal-phalangeal joint. Skin and largest lesion are 18 mm thick. Lesion is a group of small abscesses 4 mm beneath the epidermis. The amount of induration between the abscesses indicates that it is a lesion of long standing. A zone of hemorrhage at its superficial border can be attributed to recent contusion. Lymphocytes, fibroblasts, budding capillaries, caseation and cal-

cification characterize the individual abscesses. Many giant-cells are present. Some have engulfed masses of calcified material.

Two smaller nodules have the same histology as the one described. No acid-fast bacteria are found. (Figure 1.)

Foreign-body pseudotubercle: A small, shot-like, cutaneous nodule on the anterior surface of the metatarsal region. The

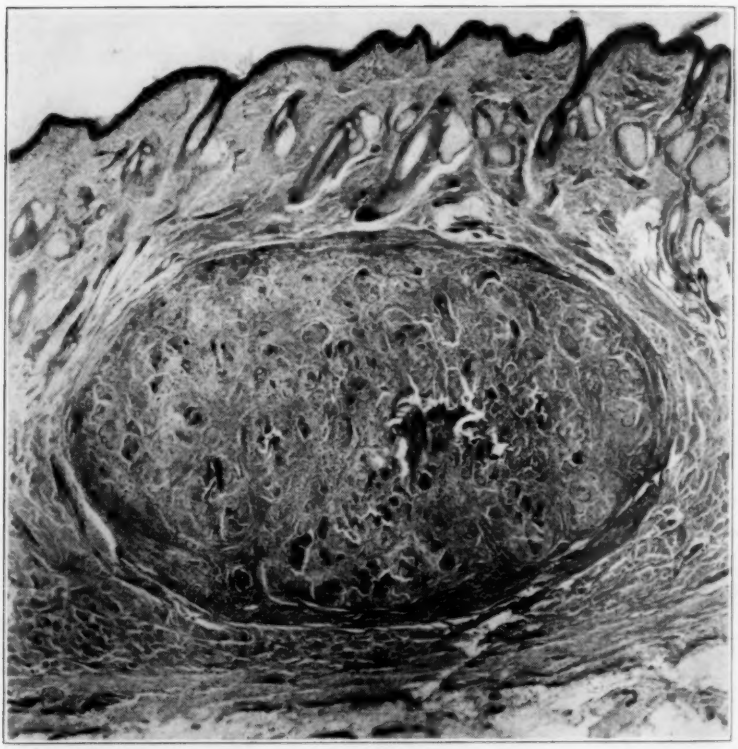

FIG. 2. Small shot-like foreign-body pseudotubercle. Its structure is quite similar to a tubercle without caseation (x 25).

skin is 3.5 mm thick. The nodule is sharply defined and flattened slightly. It measures 2 by 3 mm with its longer axis parallel to the surface of the skin. It lies immediately beneath the hair follicles and encroaches upon them slightly. The most conspicuous thing about the nodule is the number, shapes and sizes of the syncytium-like giant-cells present. Many of them enclose irregularly shaped particles and masses of material which stain only slightly, and usually not at all with hematoxylin. Fibroblasts, endothelial cells, lymphocytes and eosinophiles fill the space between the giant-cells. Occasionally a foreign body not engulfed by a giant-cell is observed. (Figures 2 and 3.)

Tuberculous abscess: A rather large, caseo-calcareous nodule about 3 by 5 cm through the longer and shorter diameters. A scar, 2 cm in diameter, on the surface of the skin directly above the nodule, is similar in appearance to a herpes tonsurans lesion. Near it is a straight scar about an inch in length.

Around the hair follicles and sebaceous glands are numerous foci of lymphocytic infiltration and a few minute young tubercles consisting of lymphocytes, endothelial cells and giant-cells. Blood-vessels extending inwardly from the cutis are surrounded with

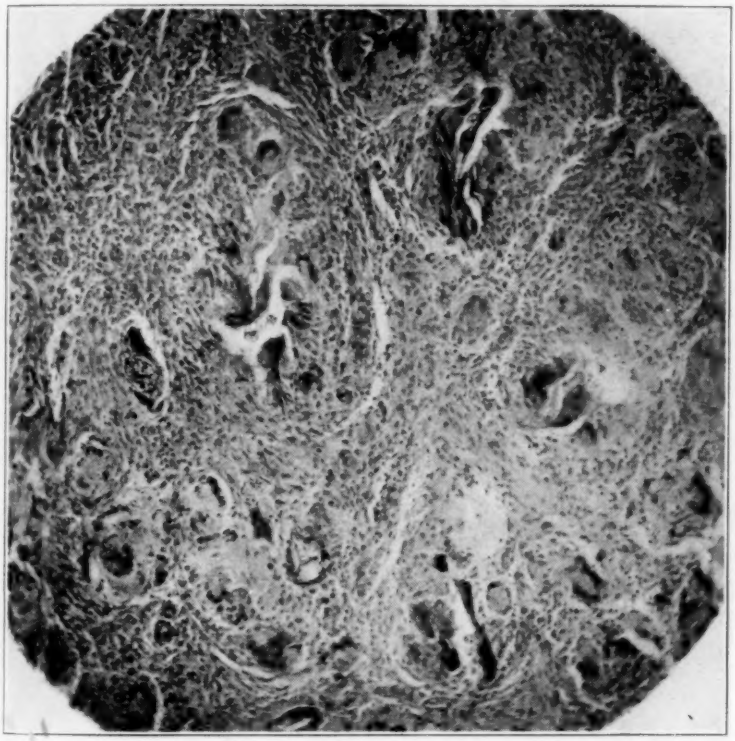

FIG. 3. Higher magnification (x 100) of a portion of the foreign-body pseudotubercle shown in figure 2. Most of the plant fibers which constitute the foreign bodies are engulfed by multinucleated giant-cells.

lymphocytes. Deeper in the subcutis are sharply circumscribed areas of pervisacular lymphocytic infiltration. In these areas the walls of old vessels frequently show hypertrophy and obliteration. Many smaller vessels form in their vicinity. As these areas increase in size, they assume the characteristics of tubercles. Endothelial cells and giant-cells appear. Central caseation and calcification next occur, and the small proliferating vessels are seen mostly near the outer border of the tubercle.

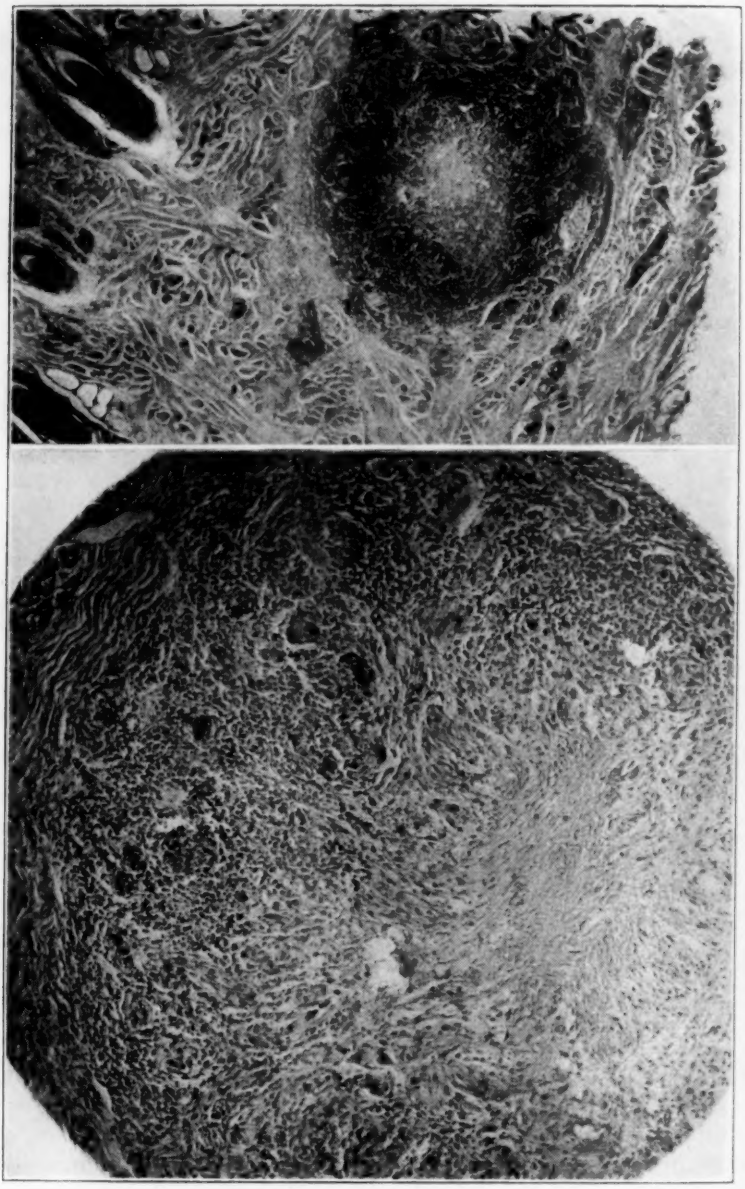

FIG. 4 (above). A young tubercle in the subcutis showing the caseous center and the zone of endothelial cells and giant-cells in which are several small blood-vessels. Note also the perivascular lymphocytic infiltration in the neighborhood of the tubercle and in the cutis (x 25).

FIG. 5 (below). Higher magnification (x 100) of a portion of the tubercle appearing in figure 4.

There is complete obliteration of the larger vessels around which the lesion had its beginning.

The greater part of the lesion consists of caseo-calcareous material. (Figures 4, 5 and 6.)

In making a differential diagnosis between pyogenic and tuberculous abscesses, difficulty was encountered in a few instances. The lesions in question appeared to be pyogenic abscesses but there was some slight evidence that they may have had a tuberculous origin. Typical of this type of nodules, which were diagnosed as pyogenic abscesses, is the following:

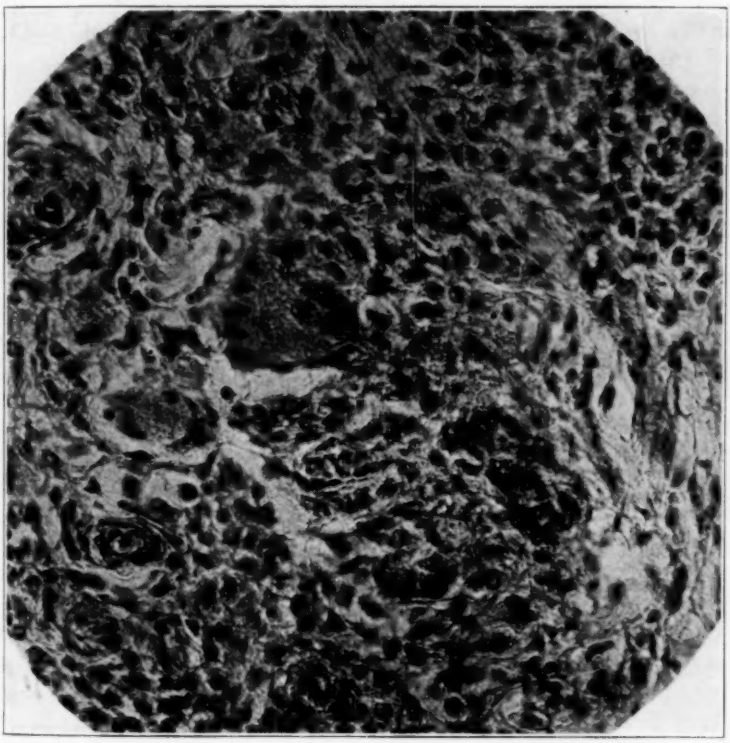

FIG. 6. A part of the same tubercle shown in figure 4, the zone of endothelial cells and giant-cells showing its vascularity (x 450).

A small nodule from the anterior surface of the upper metacarpal region. Consists of caseo-calcareous abscesses situated in the subcutis. Only the outer portion of the deepest abscess is included in any of the sections. Directly above it, but also lying in the subcutis, is a similar abscess. In close proximity to the latter are several small, caseous abscesses of uniform size

located in the corium or derma. Accessory skin structures have disappeared above these. The thickness of the skin is 6 mm.

The cellular structure surrounding the necrotic centers consists of lymphocytes and fibroblasts. Occasional multinucleated, syncytium-like giant-cells are seen in the second abscess mentioned. At the border of the abscesses and around the adjacent blood-vessels there is an infiltration with eosinophiles and lymphocytes.

COMMENT

At the beginning of this study it seemed reasonable to suppose that cattle originating in Arizona and New Mexico might have cactus-spine pseudotubercles in their skin but this investigation did not confirm this supposition. The federal veterinary inspectors in that region state that they believe such nodules are extremely rare.

The lesions diagnosed as tuberculosis by investigators in Quebec, Montana and New York were quite similar in their histopathology to the California lesions diagnosed as lymphangitis due to an acid-fast organism. Goldberg[7] made a comparative study of some similar, if not identical, California cases, several New York cases diagnosed bacteriologically and histopathologically as tuberculosis by him and Carpenter, and some cases of lupus vulgaris in man. He found them to be practically identical. He noted that these lesions differ from a typical tuberculous nodule in such organs as the spleen, lymph-glands and lungs, in that the young lesion starts its development around a blood-vessel and because of the production of many blood-vessels in the peripheral zone of the nodule. In connection with the present study ten cases of lupus in man were studied. The tubercles in lupus were similar to those found in the cutis and subcutis of cattle, except that they were avascular, they did not have their origin around blood-vessels, and they appeared to be more malignant.

In the tuberculous nodules of cattle the infection appears to enter around the hair-follicles and sebaceous glands, perhaps as a result of trauma, and follows the blood-vessels to the subcutis. Only a few completely developed tubercles were observed in the cutis. Evidently it is not a favorable place for their development. On the other hand, however, areas of lymphocytic infiltration were common in the cutis and a direct connection between them and the tubercles in the subcutis could be traced.

This view of the origin of the tubercle in the hide of animals is contrary to the view of the genesis of tuberculosis of the skin of humans recently announced by Kren and Lowenstein.[15] Their researches indicate that the origin of certain tuberculosis of the skin and of tuberculides is ascribable to the circulation of living

tubercle bacilli in the blood. Likewise, Jadassohn[16] and Ehrmann[17] believe that lichen scrofulosorum, another type of human cutaneous tuberculosis, is of hematogenous origin. Calmette,[18] on the other hand, in describing the genesis of a lupus nodule, indicates that the infection is of exogenous origin. This appears to be the case also in subcutaneous tuberculosis of cattle. The possibility of infection in this manner has been emphasized by Day.[9]

Caseation and calcification were found alike in the pyogenic abscesses and in the tuberculous nodules. There is a possibility that the pyogenic abscesses which present these changes which are characteristic of tuberculosis may originally have been tubercles.

SUMMARY

Lesions from 50 animals from six widely separated sections of the United States and one from Canada were studied. Eighteen animals came from Michigan, four from Arizona, eight from California, one from Quebec, five from Montana, three from New York and eleven from Iowa.

Practically all of the lesions in this study were located on the extremities, except three that were removed from teats. As far as is known, there was no involvement of lymph-glands in the neighborhood of the nodules. Traum states, however, that the California lesions take the formation of nodular and corded masses along the course of the lymphatics.

Among 22 lesions from the 18 Michigan cases nine nodules were tuberculous. Three were foreign-body pseudotubercles, the foreign bodies for the most part being fibres of plant origin, possibly thorns, briars, or splinters of wood. In structure, these nodules resemble closely the foreign-body pseudotubercles in man described by Warthin.[19] Cactus spines were responsible for the pseudotubercles he studied. Another of the Michigan nodules had the appearance of having been caused by a cutaneous, animal parasite, possibly one of the acarids, although no parasites were seen in the tissue. Nine cases yielded nine pyogenic abscesses in various stages of development.

Summarized briefly, this histopathological study of skin lesions of 50 tuberculin-reacting cows shows that:

1. Nodules involving the cutis of cattle are rare, but when they occur in the subcutis there is usually a pronounced inflammatory reaction in the cutis.

2. When tuberculous nodules develop in the subcutis there appears to be a direct connection between them and areas of lymphocytic infiltration around the hair-follicles, sebaceous glands and vessels in the cutis. The connection is indicated by perivas-

cular lymphocytic infiltration in the connective tissue between the two affected areas.

3. Caseation and calcification are apparently of common occurrence in subcutaneous nodules of cattle.

4. Tuberculous nodules in the hides of cattle differ from lupus of man not only in location within the skin itself, but also in their perivascular origin, their peripheral vascularity, and extensive caseation and calcification.

5. There appears to be no difference in the histopathology of the subcutaneous nodules diagnosed bacteriologically as tuberculosis, and as lymphangitis.

6. Table I gives a classification of the nodules according to their histopathology.

7. Table II gives the results of an examination of acid-fast-stained sections from 17 specimens representing the three principal types of lesions studied.

TABLE I—*Classification of nodules according to their histopathology.*

GEOGRAPHICAL DISTRIBUTION	ANIMALS FROM WHICH LESIONS WERE OBTAINED	LESIONS	DIAGNOSIS			
			TUBERCULOSIS	FOREIGN-BODY PSEUDOTUBERCULOSIS	PYOGENIC ABSCESSES	ACARIASIS
Michigan	18	22	9	3	9	1
Arizona	4	6	0	0	6	0
California	8	8	8	0	0	0
Quebec	1	1	1	0	0	0
Montana	5	5	5	0	0	0
New York	3	3	3	0	0	0
Iowa	11	11	11	0	0	0
Totals	50	56	37	3	15	1

CONCLUSION

1. The gross appearance of subcutaneous nodules cannot be entirely relied upon in making a differential diagnosis of pyogenic abscesses, foreign-body pseudotubercles and nodules now diagnosed as tuberculosis and lymphangitis.

2. In some cases a differentiation between the microscopic appearance of an old tuberculous abscess and an old pyogenic abscess of the subcutis is difficult. It is quite possible that the former infection is supplanted by the latter and as a consequence the structure of the tubercle is lost.

3. The tubercle of the disease designated as lymphangitis in California cannot be differentiated histologically from the

tubercle called tuberculosis in some other parts of the country and Canada.

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TABLE II—*Results of examination of sections and pus-smears for acid-fast bacteria.*

SPECIMEN		DIAGNOSIS OF HEMATOXYLIN-EOSIN SECTIONS	ACID-FASTS	
			SECTIONS	SMEARS
Michigan	9	Tuberculosis and pyogenic abscess	+	Not examined
	10	Pyogenic abscess	−	Not examined
	11	Pyogenic abscess	−	Not examined
	16	Pyogenic abscess	−	Not examined
	17	Foreign-body pseudotubercle and pyogenic abscess	−	Not examined
Montana	1748	Tuberculosis	+	Not examined
	1774	Tuberculosis	+	Not examined
	1782	Tuberculosis	+	Not examined
Iowa	8030	Tuberculosis	+	Not examined
	8031	Tuberculosis	+	Not examined
	8032	Tuberculosis	+	Not examined
	8070	Tuberculosis	+	+
	8071	Tuberculosis	+	+
	8072	Tuberculosis	−	−
	8073	Tuberculosis	+	+
	8074	Tuberculosis	+	−
	8088	Tuberculosis	+	−

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BUREAU TRANSFERS

Dr. Arthur J. Burley (Corn. '05) from Sheridan, Wyo., to Valentine, Neb., on field inspection.

Dr. J. H. Shoemaker (Ind. '18) from Helena, Mont., to Cheyenne, Wyo., on field inspection.

Dr. Wm. A. Cornell (K. C. V. C. '17) from Sioux City, Iowa, to Omaha, Neb., on meat inspection.

Dr. J. A. Whealy (St. Jos. '15) from Sioux Falls, S. Dak., to Seattle, Wash., on meat inspection.

Dr. C. R. Behler (Chi. '04) from Sacramento, Calif., to Kansas City, Mo., on meat inspection.

Dr. John M. Dickson (Chi. '10) from Kansas City, Mo., to Chicago, Ill., on meat inspection.

Dr. Gustave A. Kay (Chi. '02) from Nebraska City, Neb., to Cedar Rapids, Iowa, in charge of meat inspection.

Dr. S. K. Nelson (McK. '17) from Albert Lea, Minn., to Philadelphia, Pa., on meat inspection.

Dr. H. A. Schneider (U. P. '06) from Frederick, Md., to Reading, Pa., on meat inspection.

DISEASES OF YOUNG LAMBS*

By HADLEIGH MARSH, *Bozeman, Montana*
Montana Veterinary Research Laboratory

When I was asked to present a paper on sheep diseases before the Section on Sanitary Science and Food Hygiene, I was somewhat at a loss as to just what ground I should attempt to cover. After consulting your Secretary, I decided to discuss some of the diseases of young lambs. I assume that this section exists primarily for veterinarians engaged in regulatory work, and the diseases which I propose to discuss are not particularly susceptible to regulatory measures. However, the control of most of these conditions is most emphatically a matter of sanitary science.

Work with sheep diseases in general is largely in the field of prevention rather than cure. We are dealing with an animal whose individual value does not justify expensive curative treatments. Sheep are generally handled in rather large numbers where preventive measures applied to the flock as a whole are very valuable in reducing losses and making the business profitable.

This is particularly true of the diseases of young lambs. The profit in a bunch of ewes depends largely on the number of healthy lambs at shipping time. Every lamb that can be saved means additional profit, and it is better business to market 100 lambs for every 100 ewes than it is to run 125 ewes to produce 100 lambs. There is often a high mortality among young lambs, and the prevention of this loss is not to any extent a question of treatment of sick animals, but is a matter of preventive measures which are largely concerned with proper sanitation.

Disease losses in young lambs occur mostly in farm flocks and in range sheep which are shed-lambed, as opposed to range sheep which lamb on grass. Where sheep are handled on the range and are lambed on grass, the losses from disease in the lambs are negligible. But when the lambing time is pushed ahead to an unnatural season, and the lambs are handled in sheds, conditions exist which are favorable to the development of various infections in the lambs during a very critical period of their existence. In order to get heavier lambs at shipping time, there has been a tendency to move the lambing season forward into February, March and April. In the northern states this early

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

lambing season means that the ewes are lambed on hay and that sheds must be used for shelter. These unnatural conditions must be offset by special attention to sanitation if disease losses are to be avoided.

The veterinarian doing any sheep practice should have a practical knowledge of the methods of handling sheep during lambing, and should be able to advise the owner intelligently as to means of preventing disease in a practical way. The principles governing disease control in lambing-sheds are the same for the farm flock as for the range outfit. The first thing to be considered is the proper location of the lambing-shed. There has been a tendency to build lambing-sheds in creek bottoms, on land only a few feet above water, where there is brush shelter and the water supply is convenient. This is a mistake, as the first requisite in a lambing-shed is dryness. The shed should be built on well-drained land somewhat above the creek bottom, even at the expense of some convenience of water supply.

A lambing-shed should be light and well ventilated. If the lambs are clean and dry, and sheltered from rain and snow and wind, plenty of fresh air will not hurt them, even in cold weather. A sheep-shed should never be closed tightly when it is full of sheep.

In most cases the lambing-shed has no floor. The ground constituting the floor of the shed should be thoroughly cleaned after lambing and, if possible, the shed should not be used for sheep until the next lambing season. During lambing, straw bedding should be used. There is a tendency to bed deeply with straw at the beginning of lambing and not clean out frequently. The result of this is that the bedding becomes saturated with the excrement of the sheep and constitutes a culture medium for pathogenic bacteria. The ground should be bedded lightly with straw and cleaned frequently. This is especially important for the individual pens in which the ewes and lambs are held.

As a rule, the ewes should not be shedded at all, but should drop their lambs on dry, well-drained land. When the ewes are on wet ground or confined in sheds at night, the wool around the udder and hindquarters becomes soiled with mud and manure, which is probably one of the sources of intestinal infection in the lambs. It is good practice in any case to "tag" the ewes about two weeks before lambing. This operation consists of trimming the wool around the udder, the inner surface of the thighs, and in the perineal region.

Diseases Due to Management of the Ewe During Pregnancy

There are at least two disease conditions which occur in young lambs which are due to deficiencies in the feed supplied to the

ewes during the gestation period. One of these diseases is goiter. In those parts of the country which are deficient in iodin, this condition may at times be the cause of a heavy loss in lambs. Many of the lambs are born dead, and those that live are weak and often very lightly wooled, and many of them show much enlarged thyroids. Such lambs usually die within the first few days after birth. Prevention of goiter is very simple in sheep, and the results are certain. The iodin deficiency in the feed and water can very easily be overcome by feeding iodized salt to the ewes during the gestation period. It has been found that the addition of 1 ounce of potassium iodid to 300 pounds of salt is sufficient to prevent goiter. We thoroughly mix an ounce of potassium iodid with two pounds of table salt, and enough charcoal to give the mixture a dark color. This is then thoroughly mixed with 300 pounds of special stock salt. The salt should be fed in troughs, and only enough put out at one time to supply sheep for a few days. If a large amount is put out, there is some loss of iodin content due to exposure to light and moisture. For this reason we do not recommend iodized block salt for goiter prevention.

The other lamb disease which may result from faulty metabolism of the ewe is the habit of chewing at dirt or wool. It is probable that a certain amount of dirt-eating or wool-eating may be considered normal for a lamb which is not out on the range where it can nibble on grass. But in certain sheep-growing areas, there is excessive dirt-eating, which results in some lamb loss due to dirt and wool impaction of the abomasum. This condition has been theoretically ascribed to a deficiency of calcium phosphate in the diet of the ewe. Clinical experience has shown that feeding bonemeal to the ewes, in their salt, has in some cases prevented excessive dirt-eating by the lambs.

Infectious Diseases

Necrobacillosis of the Liver

For the first few hours after the birth of the lamb, the stump of the umbilical cord affords an excellent culture medium for the development of bacteria with which the cord may come in contact on dirty bedding or on the ground in corrals and sheds. Umbilical infection does not often result in a visible local lesion, but is manifested in several ways when the lambs are from one to three weeks old.

One of the commonest conditions resulting from umbilical infection is necrobacillosis of the liver. The usual history of losses from this cause is that the lambs die when they are from one to three weeks old, without showing any characteristic symptoms.

Autopsy of these cases shows the characteristic lesions in the liver. There are usually a number of necrotic areas in the liver, appearing on the surface as rather sharply circumscribed yellowish grey areas, which are found on section to consist of a somewhat caseous necrotic material. Smears from the borders of these necrotic areas showed that the predominant organism present is the *Actinomyces necrophorus*.

This type of umbilical infection is strictly a product of shed-lambing as opposed to grass-lambing. It is generally considered to occur more commonly where old sheds that have been used for horses and cattle are converted into lambing-sheds. It may, however, occur in new lambing-sheds built on clean ground, particularly if the land is low and moist. I have observed a heavy loss from this cause in a new lambing-shed, which was bedded with straw at the beginning of the lambing season and never cleaned. Under these conditions the straw became saturated with the excrement of the sheep, constituting an excellent medium for the development of the causative organism.

It has been found that this condition can definitely be prevented by proper disinfection of the umbilical cord within the first few hours after birth, or while the cord is still moist. The most satisfactory antiseptic to use is tincture of iodin. In order to insure results, the stump of the cord and the skin immediately surrounding the umbilicus must be bathed in the solution. To accomplish this, we cut off the cord, leaving a stump of about 1½ inches, and then dip the stump in iodin contained in a small, wide-mouthed container of about 3 to 4 ounces capacity. The neck of the container is pressed against the abdomen of the lamb and the lamb is then tipped back so that the iodin comes in contact with the whole region. For this purpose tincture of iodin has advantages over other disinfectants in that it is penetrating and astringent, and the yellow stain shows whether the work has been done thoroughly or not.

Posterior Paralysis

Lambs from two weeks to one month old may develop a posterior paralysis. Such lambs as a rule do not show evidence of a systemic disturbance, as they appear bright and have a good appetite, but their hind legs become completely paralyzed. The paralysis in these cases is due to a focal infection of the spine, with the formation of a lesion which causes pressure on the cord. The infection in these cases is not specific. In one case which we observed, the spinal lesion was due to *A. necrophorus* infection, and in others staphylococci have been the causative organisms. The avenue of infection is in some cases the umbilicus and in others the docking wound.

ARTHRITIS

In common with the young of other species of domestic animals, lambs may develop arthritis as a result of exposure of the umbilicus, castration wound or docking wound to infected bedding or ground.

In one type of arthritis the lameness and enlarged joints appear very early in life, usually during the first month. These acute cases are found to be due to infection with pyogenic bacteria, and are presumably the result of umbilical infection in most cases. Clinical experience shows that the occurrence of such cases can be prevented by disinfection of the umbilical region at birth.

Another type of arthritis is of a more chronic nature, and is a specific infection with *Erysipelothrix rhusiopathiae*, the bacillus of swine erysipelas. The symptoms in these cases may appear at any time from the end of the first week of life to the fourth or fifth month. When the symptoms appear in lambs under one month of age, there is first an acute lameness in one or more legs. The acute stage may persist for a few days or a week, after which there is marked improvement, with a complete disappearance of symptoms in some cases. This apparent recovery in many cases is not real, as several weeks or months later it is noticed that the lamb is not growing properly and he may again develop lameness which becomes chronic. In the range country this condition appears on the summer range as causing "stiff lambs," which are unable to follow the bands at shipping time.

The lesions in this type of arthritis are not suppurative. In most cases the enlargement of the joints is not sufficient to be noticeable during life. There is an inflammation and thickening of the joint capsule, with a moderate amount of a gray fluid exudate. In some cases there is erosion of the articular surfaces of the bones. The joints most frequently affected are the carpal, tarsal and femoro-tibial.

The avenues of infection are the umbilicus soon after birth, the castration wound, and the docking wound. It has been clinically possible to prevent this disease by disinfecting navels and docking with proper precautions. We have had the best results by docking with a hot iron and turning out on clean grass. Docking with an emasculator has been practiced to some extent, and circumstantial evidence indicates that infection with the arthritis organism (*E. rhusiopathiae*) occurs to a greater extent in lambs docked with the emasculator than in those docked with the hot iron. This may be due to the fact that the emasculator leaves a certain amount of crushed dead tissue in which bacteria may multiply.

Blackleg and Tetanus

The docking and castration wounds constitute portals of entry for various infections, particularly with pathogenic anaerobes. Two diseases caused by this class of organisms which occasionally cause heavy losses are blackleg and tetanus. We have known of several instances where many lambs died of blackleg, as the result of being turned, after docking, into a pasture where cattle had recently died of blackleg. We have also seen tetanus develop as a result of holding freshly docked lambs in dirty corrals.

Dysentery

In the shed-lambed range bands of the northwestern states, there occurs at times during the first few days of life of the lambs a severe diarrhea which is apparently of the same nature as white scours of calves. This diarrhea usually develops within the first forty-eight hours. In some outbreaks the mortality is quite high, the fatal cases living only from one to three days. The lambs are rapidly prostrated. The fecal material becomes fluid and putty-colored, and has a fetid odor. The milder cases have a light yellow diarrhea and are not so completely prostrated. These cases recover, but rather slowly.

This disease is evidently the result of bacterial infection, but no specific organism has as yet been definitely incriminated. It is evident that two important factors are cold storms and filth. When the weather is warm and dry, the resistance of the lambs is so great that the infection can not gain the upper hand. When the new-born lamb's resistance is lowered by cold wet storms, intestinal bacteria may become pathogenic. These same weather conditions also increase the amount of filth, to which the lamb is exposed, on the udder and wool of the ewe.

As preventive measures we therefore advocate sanitary measures, such as keeping the shed and corrals clean and dry and keeping the ewes' udders and thighs clean. This latter object is accomplished by "tagging" the ewes before lambing, and keeping the drop band on dry, well-drained ground. Care should be taken also to prevent exposing the lambs to severe stormy conditions. There is some indication that calf scour serum may help, if given at birth as a preventive measure. Intestinal antiseptics, such as sulfo-carbolates, appear to have value as treatment.

In shed-lambing, it is the general practice for the lamber to suckle each lamb before leaving it. This operation may be repeated if the lamb does not appear to be as active as he should. In sheds where scours is occurring, this practice probably spreads

the infection. A lamber handling scouring lambs should not be allowed to handle healthy lambs until his hands have been cleaned.

Pneumonia

There occurs at times a small percentage of loss, among lambs in the first week of life, due to pneumonia. In the cases of this kind which we have investigated, the pneumonia was evidently the result of a Pasteurella infection. This seems to be a disease of farm flocks, and the bacteria are probably present in dirty sheds, and become pathogenic as a result of lowered resistance of the lambs due to bad weather during early lambing. In several cases owners of small flocks have used hemorrhagic septicemia aggressin soon after birth, and have apparently prevented the development of this type of pneumonia.

Miscellaneous Diseases

Stiff Lambs or White-Muscle Disease

A condition which has been observed in farm flocks, both in the eastern and western parts of the country, produces what the owners describe as stiff lambs. The disease appears when the lambs are about one month old, often coincident with turning the lambs out of sheds into pastures. The symptoms are a peculiar stiffness of gait, and difficulty in rising from a recumbent position. The appetite remains good and the lambs show little evidence of systemic disturbance. However, a majority of the affected lambs die, as a result of inability to follow the bunch and obtain nourishment.

The lesions found at autopsy are very striking. There is a degeneration of certain groups of muscle fibers, which show as white areas. The distribution of these areas is bilaterally symmetrical. That is, if a certain muscle in the thigh of the right leg shows such an area, the corresponding muscle of the left leg will show a similar area. The lesions occur in the muscles of the limbs and back, and also are found frequently in the heart muscle. It has been determined that the white appearance of these groups of muscle fibers is due to a deposit of calcium phosphate. Chemical analysis of muscles containing these areas shows a marked increase in the amount of calcium over that found in normal muscle and a smaller increase in phosphorus. This indicates that the deposit is the insoluble tricalcium phosphate, and that there is in these lambs a disturbance of the normal calcium and phosphorus balance.

The cause of this abnormal mineral metabolism has not yet been determined. In Montana it occurs only in farm flocks in certain limited areas, and experiments are now being carried

on in an attempt to determine what conditions exist in these areas which may cause the disease.

Conjunctivitis

Conjunctivitis in young lambs occurs quite frequently. In some cases this is an infectious ophthalmia, which affects many of the lambs in a flock and may be treated by the usual methods of treating conjunctivitis.

Another form of conjunctivitis which is more common but affects a smaller percentage of a flock is due to entropion. This is a condition which may be hereditary, as its distribution is irregular, and as it apparently occurs more frequently in black-faced lambs than in other breeds. In these cases the lower eyelid is longer than it should be, and entropion results, causing irritation of the conjunctiva. The development of a severe conjunctivitis can be prevented by a simple procedure which is very successful and which avoids surgical interference. A small amount of the excess skin is picked up with forceps or the fingers, and tied off with linen thread. No further attention is necessary, as the excess tissue sloughs away and the thread drops off. In a large purebred outfit, where a considerable percentage of the new-born lambs show entropion, this operation is performed regularly by the lambing foreman during the first two days of life of the lambs.

The diseases briefly discussed in this paper are the most important pathological conditions with which we have to deal in lambs during the first month of life. There are, of course, other conditions which may require diagnosis and treatment, but those mentioned are the diseases which have come to our attention as causing the major losses. Considerable losses of young lambs occur at times where no specific disease is found, and the deaths are due to undernourishment. In this connection, we sometimes see lambs showing a dropsical condition, which apparently is a result of malnutrition. We have also observed an infectious gastro-enteritis in lambs under one month of age, and in some of these cases the *Clostridium edematis* has been incriminated as the causative organism. The parasitic diseases do not cause trouble until a little later in life. A diarrhea due to coccidiosis is sometimes observed in lambs not more than one month old. In our experience, however, coccidiosis in young lambs is not a serious condition, as it tends to clear up spontaneously after two or three weeks.

It will be seen that from the standpoint of disease, the reduction of infant mortality in lambs, as in the human race, is largely a matter of proper diet for the mother, and proper sanitation for the new-born lamb.

FURTHER OBSERVATIONS ON THE CONTROL OF CONTAGIOUS ABORTION BY MEANS OF BLOOD-TESTING AND SEGREGATION*

By I. E. NEWSOM *and* FLOYD CROSS

Section of Veterinary Pathology, Colorado Agricultural Experiment Station, Fort Collins, Colorado

Since our previous publication,[1] the control of Bang's disease by means of blood-testing and segregation has become so widespread that thousands of herds are now either free or are in process of becoming so. Publications are so numerous that compiling a bibliography on abortion disease becomes a herculean task. We may be pardoned, therefore, if we give few references. It is not the intention of the authors to clutter up the literature with the mere record of another herd but to attempt to throw additional light on certain points that still serve to complicate the procedure.

In March, 1927, this section was appealed to by the United States Veterans Bureau hospital at Fort Lyon, Colorado, to offer advice in connection with the abortion problem in their dairy herd. The first test was made on March 25 of that year, at which time 35 reactors were removed from a herd totaling 125 animals of breeding age. The two herds were kept in separate pens and pastures, separated by only a few feet and milked in the same barn. The positive cows were admitted to the barn through a separate door and were always milked after the negative animals had been released. The drainage was away from the negative herd.

The superintendent of the hospital was a physician who took great interest in the work, and the foreman of the herd was an agricultural college graduate of more than the average intelligence. The whole set-up seemed ideal for a rapid elimination of the disease. Tests were made every 60 days.

The last positive animals were removed from the herd exactly three years after the work was begun. Eighty-three positive animals were taken out and only eight were returned.

The negative herd (table I) comprised 90 animals at the beginning of the work, dropped as low as 69 on the fourteenth test, and at the last test was composed of 106. The positive herd originally had 35 reactors but was gradually depleted by sale and slaughter until the twenty-third test, when it passed out of existence.

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The total number of abortions was 45. Of this number, 19 had taken place prior to the application of the first test. All but two were positive reactors on the first test and these two remained negative. Twenty-six abortions occurred later. Of these, 15 were in animals positive at the time of the abortion and six in those that became positive afterward, leaving five that remained negative. Thus only seven of the whole 43 could be said to be truly negative abortions, that is, not due to the Bang organism. Two cows aborted twice, so that the number of aborting animals was 43.

TABLE I—*Number of animals in the positive and negative herds at the different tests.*

TEST	DATE	POSITIVE	NEGATIVE
1	3–25–27	35	90
2	5–25–27	34	86
3	7–25–27	36	80
4	9–24–27	34	93
5	12–10–27	24	88
6	2–11–28	21	79
7	4– 7–28	24	78
8	6–15–28	24	74
9	8–18–28	26	74
10	10–27–28	27	90
11	1– 5–29	24	85
12	3– 9–29	31	76
13	4–19–29	29	74
14	6– 4–29	25	69
15	7– 9–29	23	83
16	8–10–29	26	77
17	9–14–29	25	80
18	10–19–29	21	83
19	12–21–29	25	82
20	3–29–30	9	77
21	4–29–30	7	74
22	5–31–30	6	84
23	7–31–30	6	87
24	11–19–30	0	89
25	8– 6–31	0	106

FIVE WAVES OF REINFECTION

The second test showed five new reactors, the third four, the fourth one, the fifth two and the sixth none. On the seventh test, however, five new reactors developed, followed by two, two and four, respectively. There were no reactors again on the eleventh test. The next three tests showed another wave of infection totaling 17 animals. At the fifteenth test there were no reactors again, but on the sixteenth and seventeenth tests four new positives were taken out. After two more negative tests, two additional positives were found at the twentieth

test. Five more tests made since that time have shown no new reactors. As approximately 14 months have elapsed since there has been a positive reactor, it now seems safe to say that the herd is clean.

Surveyed in retrospect it looks as though there had been five distinct waves of reinfection. We explained the first one on

TABLE II—*Negative cows aborting in the clean herd in relation to the reactors.*

TEST	DATE	REACTORS	COW	ABORTED	BECAME POSITIVE
1	3–25–27	35			
2	5–25–27	5			
			549	7– 4–27	Never
3	7–25–27	4			
			117	7– 7–27	Never
4	9–24–27	1			
			641	11– 3–27	12–10–27
5	12–10–27	2			
6	2–11–27	0			
7	4– 7–28	5			
8	6–15–28	2	833	7–28–28	8–18–28
9	8–18–28	2	(707)	9–13–28	4–19–29
10	10–27–28	4	(719)	9–19–28	6– 4–29
11	1– 5–29	0			
12	3– 9–29	8			
			726	4–16–29	4–19–29
13	4–19–29	5			
14	6– 4–29	4			
15	7– 9–29	0			
16	8–10–29	3			
17	9–14–29	1	722	8–28–29	Never
18	10–19–29	0	650	11–14–29	3–29–30
19	12–21–29	0			
	3–29–30	2			
20	4–29–30	0	676	4–20–30	Never
21					
22	5–31–30	0			
23	7–31–30	0			
24	11–19–30	0			
			728	4– 6–31	Never
25	8– 6–31	0			

the finding that the manure from the infected pens was thrown onto the spreader, after which it was driven into the clean corral, where, while being loaded, the cattle gathered around it and picked out straw. This practice was stopped. The second wave was thought to be due to turning the clean cattle onto a pasture where the infected manure had been spread. By the

time the third wave came all such explanations were exhausted and it was concluded that it was impossible to maintain the infected herd so near the clean one. Accordingly, on May 20, 1929, the positive reactors were moved to another farm about a mile away. Following this radical procedure, four new animals reacted on the sixteenth and seventeenth tests and two more on the twentieth.

The herd manager then called attention to the fact that negative aborters were becoming positive after their isolation period and after their return to the clean herd. Table II, which shows the relationship of the aborting animals to these waves of infection, is very illuminating. It shows that cow 641, after having passed four clean tests, aborted in the clean herd on November 3, 1927, was immediately isolated and became positive on the next test taken December 10, 1927. She might well have caused the flare-up beginning on the seventh test, April 7, 1928, five months after her abortion.

Cow 833, after having passed eight clean tests, aborted on July 28, 1928, and was positive on the next test, August 18, 1928. Premonitory signs of abortion were discovered and she actually expelled the fetus in isolation. There is a possibility that she expelled some organisms before being isolated, as three months later there were four new reactors. Neither of these animals was returned to the clean herd, as both were positive on their next test.

Cows 707 and 719 are still further interesting, as they aborted in September, 1928, after having passed several negative tests. Both were immediately isolated, the fetus and membranes cleaned up and the places disinfected. Both were returned to the clean herd on November 26, 1928, after having passed two clean tests. Cow 707 became positive on April 19, 1929, seven months after she aborted and after having passed four clean tests subsequent to that event. Cow 719 passed three more tests after returning to the clean herd and finally became positive on June 4, 1929, nine months after aborting.

Whether the abortions of these two animals in September, 1928, caused the 17 reactions of the twelfth, thirteenth and fourteenth tests or whether their return to the herd was responsible it is impossible to state. If the first view be held, then the incubation period was about six months; if the latter, then it was about three months. The previous experience with cows 641 and 833 would indicate that it was the abortion, as those two animals were never returned. It ought to be pointed out that cows 707 and 719 themselves reacted on the thirteenth and fourteenth tests, respectively, but of course they did not in-

TABLE III—*Cows that aborted before tests were started.*

Cow		637	622	565	534	101	574	667	992	533	991	994	105	106	606	108	570	109	603	846
ABORTED	1925					9–28	1–6		8–26											
	1926	11–18	11–?	10–29	4–29		10–14	2–8		5–24	9–11	6–26	10–18	5–24	11–1	5–27	7–22	?	8–13	?
TEST	DATE	REACTIONS																		
1	3–25–27	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	− − −	− − −
2	5–25–27	+ + −		+ + +	+ + +	+ + +		+ + +	+ + +	+ + +	+ + +	+ + ±			+ + +	+ + +	+ + +		− − −	− − −
3	7–25–27	+ − −		+ + −	+ + ±	+ + +		+ + ±	+ + +	+ + +	+ + ±	+ + ±			+ + +	+ + ±	+ + −	+ + +	− − −	− − −
4	9–24–27	+ + −		+ + +	+ + +	+ + +		+ + ±	+ + +	+ + +	+ + −	+ ± −			+ + +	+ −	− − −		− − −	− − −
5	12–10–27	+ + +			+ + ±			+ + +	+ + +		+ − −	+ − −			+ − −		+ − −		− − −	− − −
6	2–11–28	+ + −			+ + +			+ ± −	+ + +		+ − −	± − −					± − −			− − −
7	4– 7–28	+ + −			+ + ±			+ ± −	+ + +		+ − −	± − −					+ − −			− − −
8	6–15–28	+ + −			+ + +			+ ± −	+ + +		+ + −	+ − −					+ − −			− − −
9	8–18–28	+ + −			+ + +			+ + −	+ + +		+ − −	+ ± −					+ − −			− − −
10	10–27–28	+ + +			+ + +			+ + +	+ + +		+ ± −	± − −					+ − −			− − −
11	1– 5–29				+ + +			+ − −	+ + +		± − −	± − −					+ − −			− − −
12	3– 9–29				+ + +			± − −	+ + +		± − −	± − −					+ − −			− − −
13	4–19–29				+ + ±			± − −	+ + +		± − −	+ − −					+ − −			− − −
14	6– 4–29							− − −	+ + +		± − −	± − −					− − −			− − −
15	7– 9–29							± − −	+ + +		± − −	± − −					± − −			− − −
16	8–10–29							+ − −	+ + +		± − −	+ ± −					± − −			− − −
17	9–14–29							− − −	+ + +		− − −	± − −					± − −			− − −
18	10–19–29							− − −	+ + +		− − −	± − −					− − −			− − −
19	12–21–29							+ + −	+ + +		− − −	± − −					− − −			− − −
20	3–29–30							+ + +			− − −	− − −					+ + −			− − −
21	4–29–30										− − −	± − −					± − −			− − −

In test 22 (5-31-30), cows 991 and 846 were negative. In test 23 (7-21-30), cows 991, 994 and 846 were negative. In test 24 (11-19-30), cows 991 and 846 were negative.

TABLE IV—*Cows positive at time of abortion.*

Cow		646	649	640	672	643	659	713	745	665	683	642	681	719	107
TEST	DATE	REACTIONS													
1	3–25–27	+ ± −	− − −	− − −	− − −	− − −	− − −			− − −	− − −	− − −	− − −		+ + +
	3–28–27														A
2	5–25–27	+ + −	− − −	− − −	− − −	− − −	− − −			− − −	− − −	− − −	− − −		+ + −
3	7–25–27	+ + ±	+ + −	+ + +	− − −	− − −	− − −			− − −	− − −	− − −	− − −		
	9–17–27		A												
4	9–24–27	+ − −	+ + +	+ + +	− − −	− − −	− − −	− − −		− − −	− − −	− − −	− − −	− − −	
	10– 3–27			A											
5	12–10–27	+ ± −	+ + −	+ + +	+ ± −	− − −	− − −	− − −		− − −	− − −	− − −	− − −	− − −	
6	2–11–28	+ − −	+ + +	+ ± −	+ + +	− − −	− − −	− − −		− − −	− − −	− − −	− − −	− − −	
	3–29–28				A										
7	4– 7–28	+ − −	+ + +	+ + +	+ + +	+ + +	− − −	− − −		− − −	− − −	− − −	− − −	− − −	
	4–19–28					A									
8	6–15–28	+ − −		+ + +	+ + +	+ + +	− − −	− − −		− − −	− − −	− − −	+ + −	− − −	
9	8–18–28	+ ± −		+ + +	+ + +	+ + +	− − −	− − −		− − −	− − −	− − −	+ + ±	− − −	
	9–19–28													A	
10	10–27–28	± − −		+ + +		+ + +	+ + ±	+ + +	− − −	− − −	− − −	− − −	+ + +	− − −	
	12–27–28	A					A								

TABLE IV—*Cows positive at time of abortion—Continued.*

Cow		646	649	640	672	643	659	713	745	665	683	642	681	719	107
TEST	DATE	REACTIONS													
11	1- 5-29	+++				+++	+++	+++	− − −	− − −	− − −	− − −	++±	− − −	
	1-14-29							A							
12	3- 9-29	+− −				+++	+++	+++	− − −	− − −	− − −	− − −	+++	− − −	
	4-13-29					A									
13	4-19-29	+− −				+++	+++	+++	± − −	− − −	− − −	− − −	+++	− − −	
14	6- 4-29	+±−				+++		+++	++−	+− −	+++	− − −	+++	+±−	
15	7- 9-29	++±				+++		+++	+++	+++	+++	− − −	+++	+±−	
16	8-10-29	+++				+++		++±	++±	+++	+++	+++	+++	+±−	
	8-25-29									A					
17	9-14-29	++±				+++		+++	+++	+++	+++	+++	+++	+++	
	9-25-29								A						
	9-29-29										A				
	10-11-29											A			
18	10-19-29					+++		+− −	+++		++−	+++		+±−	
	12- 6-29												A		
19	12-21-29	+++						+++	++−	+++	++−	+++	+++	+− −	
	2- 1-30													A	
20	3-29-30									+++	+++	+++	+++		
21	4-29-30										+++	+++	+++		
23	7-31-30										+++	+++	+++		

A=aborted.

fect themselves. The presumption would have to be that they were infected by either 641 or 833.

Cow 726 is very similar to cows 641 and 833, as she aborted in the clean herd on April 16, 1929, and became positive three days later. In spite of the usual precautions she seems to have infected four other cattle that reacted some four months later.

Cow 650 is another one similar to 707 and 719, in that she was isolated just prior to the abortion, passed two negative tests and was returned to the clean herd after a 60-day interval. She was positive on the next test after returning to the herd, four and one-half months after her abortion. Since she herself was one of the reactors on the twentieth test, only one infected animal can be attributed to her. It is even possible that the other was a holdover from a previous infection, the same as 650 herself.

Aborters 549, 117, 722, 676 and 728 are placed in table II but no spread is attributed to them since they were never positive, either before or after the abortion.

Summarizing this portion of the work, it may be said that of the 11 abortions occurring in the clean herd or immediately after isolation, six of the responsible animals became positive in from three days to nine months after aborting and five remained negative. The five waves of reinfection seem to have been due to previously negative cows aborting either in the clean herd or immediately after isolation.

Cows Aborting Before Starting the Tests

Table III gives the reactions of the cows whose history indicated that they had aborted before the tests were started.* Out of the 19 animals in this group, all but two were positive on the first test and these two continued negative as long as they were tested. One of them was tested 24 times. Only one of the positive animals (cow 991) ever became negative for a sufficient length of time to be considered safe to be returned to the clean herd.

Cows Positive at the Time of Abortion

In table IV will be seen the reaction history of the animals that were positive at the time they aborted. There were 14 in this group. None ever became negative during the time they were under observation. Two of them (cows 643 and 719) aborted twice.

*All tests were run at three dilutions, 1:50, 1:100 and 1:200, and appear in this order in the tables.

Cows Negative at the Time of Abortion and Becoming Positive Later

Six cows comprised this group. These animals are more completely discussed in connection with the waves of reinfection for which they seemed to be responsible. Their blood reactions are given in table V. These are the dangerous animals in any herd

Table V—*Cows negative at time of abortion and becoming positive later.*

Cow		641	833	650	707	719	726
Test	Date	Reactions					
1	3–25–27	− − −	− − −	− − −			
2	5–25–27	− − −	− − −	− − −			
3	7–25–27	− − −	− − −	− − −			
4	9–24–27	− − −	− − −	− − −	− − −	− − −	
	11– 3–27	A + − −					
5	12–10–27	+ + −	− − −	− − −	− − −	− − −	
6	2–11–28	+ + −	− − −	− − −	− − −	− − −	
7	4– 7–28	+ ± −	− − −	− − −	− − −	− − −	− − −
8	6–15–28	+ + −	− − −	− − −	− − −	− − −	− − −
	7–28–28		A				
9	8–18–28	+ + −	+ + +	− − −	− − −	− − −	− − −
	9–13–28				A		
	9–19–28					A	
10	10–27–28	+ + +	+ + +	− − −	− − −	− − −	− − −
11	1– 5–29	+ + +	+ + +	− − −	− − −	− − −	− − −
12	3– 9–29	+ + +	+ + +	− − −	− − −	− − −	− − −
	4–16–29						A
13	4–19–29		+ + +	− − −	+ ± −	− − −	+ + +
14	6– 4–29		+ + +	− − −	+ + ±	+ ± −	
15	7– 9–29		+ + +	− − −		+ ± −	
16	8–10–29		+ + +	− − −		+ ± −	
17	9–14–29			− − −		+ + +	
18	10–19–29			− − −		+ ± −	
	11–14–29			A			
19	12–21–29			− − −		+ − −	
	2– 1–30					A	
20	3–29–30			+ + +			
21	4–29–30			+ + +			
23	7–31–30			+ + +			

A = aborted.

where eradication is under way. In this herd every such animal seems to have produced some reinfection. Apparently this was true whether they actually discharged the fetus in the clean premises or were isolated immediately before the abortion took place.

Cows Negative at Time of Abortion and Remaining Negative

Only five animals are in this group. Cows 549 and 117 were returned to the clean herd approximately 60 days after aborting.

Cow 549 remained there three months, and cow 117 for five months before they were sold. Cow 722 remained in isolation four months, was then returned to the clean herd and is still there, two years after the abortion. Cow 676 was isolated for seven months and has now been in the clean herd for about nine months. Cow 728 aborted in April, 1931, and in September.

TABLE VI—*Cows negative at time of abortion and remaining negative.*

Cow		722	549	117	676	728
TEST	DATE	REACTIONS				
1	3-25-27		– – –	– – –	– – –	
2	5-25-27		– – –	– – –	– – –	
	7- 4-27		A			
	7- 7-27			A		
3	7-25-27		– – –	– – –	– – –	
4	9-24-27		– – –	– – –	– – –	
5	12-10-27		– – –	– – –	– – –	
6	2-11-28				– – –	
7	4- 7-28	– – –			– – –	
8	6-15-28	– – –			– – –	
9	8-18-28	– – –			– – –	– – –
10	10-27-28	– – –			– – –	– – –
11	1- 5-29	– – –			– – –	– – –
12	3- 9-29	– – –			– – –	– – –
13	4-19-29	– – –			– – –	– – –
14	6- 4-29	– – –			– – –	– – –
15	7- 9-29	– – –			– – –	– – –
16	8-10-29	– – –			– – –	– – –
	8-28-29	A				
17	9-14-29	– – –			– – –	– – –
18	10-19-29	– – –			– – –	– – –
19	12-21-29	– – –			– – –	– – –
20	3-29-30	– – –			– – –	– – –
	4-20-30				A	
21	4-29-30	– – –			– – –	– – –
22	5-31-30	– – –			– – –	– – –
23	7-31-30	– – –			– – –	– – –
24	11-19-30	– – –			– – –	– – –
	4- 6-31					A
25	8- 6-31	– – –			– – –	– – –

A = aborted.

She is still in isolation although she has passed four clean tests since aborting (table VI).

The question arises as to the time that must elapse before a negative aborter can safely be returned to the clean herd. The experience with cow 719 (table I), indicates that nine months is not too long a time and raises the point whether as a practical procedure it is ever wise to return an aborting animal, in other words, whether it would not be better to sacrifice a few animals

that would remain negative than to run the risk of their becoming positive.

The previous experience of the authors leads to the conclusion that after the herd is once clean, negative aborters may be safely returned after a 30-day isolation period but that during the clean-up all aborters may well be eliminated.

Cows Positive Transferred to the Clean Herd

In this group of eight animals cow 753 may be disregarded, since she showed only an incomplete reaction on one test and never should have been isolated. Cow 834 never had a titre above 1:50 and cows 995, 993 and 424 showed only one incomplete as high as 1:100. Cow 850 had a single doubtful at 1:200, leaving only two animals that were ever complete in all three

Table VII—*Positive cows transferred to clean herd.*

Cow		834	618	995	850	424	993	991	753
Test	Date	Reactions							
	9-11-26							A	
1	3-25-27	+ − −	+ + +	− − −	− − −	− − −	− − −	+ + +	
2	5-25-27	± − −	+ + −	+ − −	+ − −	+ − −	− − −	+ + +	
3	7-25-27	± − −	+ + ±	+ ± −	+ + ±	+ − −	+ ± −	+ + ±	
4	9-24-27	± − −	+ − −	+ − −	+ − −	+ − −	+ − −	+ + −	
5	12-10-27	− − −	± − −	+ − −	+ − −	+ − −	+ − −	+ − −	
6	2-11-28	− − − *	− − −	+ − −	± − −	± − −	+ − −	+ − −	
7	4- 7-28	− − −	± − −	± − −	− − −	± − −	+ − −	+ − −	
8	6-15-28	− − −	− − −	− − −	− − −	+ − −	+ − −	+ + −	
9	8-18-28	− − −	− − −	− − −	− − −	± − −	± − −	+ − −	
10	10-27-28	− − −	− − −	− − − *	− − − *	+ ± −	− − −	+ ± −	
11	1- 5-29		− − −	− − −	− − −	± − −	− − −	± − −	
12	3- 9-29		− − − *	− − −	− − −	± − −	− − − *	± − −	± − −
13	4-19-29	*	− − −	− − −	− − −	− − −	− − −	± − −	− − −
14	6- 4-29		− − −	− − −	− − −	− − −	− − −	± − −	− − −
15	7- 9-29		− − −	− − −	− − −	− − −	− − −	± − −	− − −
16	8-10-29		− − −	− − −	− − −	− − − *	− − −	± − −	− − − *
17	9-14-29		− − −	− − −	− − −	− − −	− − −	− − −	− − −
18	10-19-29		− − −	− − −	− − −		− − −	− − −	− − −
19	12-21-29		− − −	− − −	− − −		− − −	− − −	− − −
20	3-29-30		− − −	− − −	− − −		− − −	− − −	− − −
21	4-29-30		− − −	− − −	− − −		− − −	− − − *	− − −
22	5-31-30		− − −	− − −	− − −		− − −	− − −	− − −
23	7-31-30		− − −	− − −	− − −		− − −	− − −	− − −
24	11-19-30		− − −	− − −	− − −		− − −	− − −	− − −
25	8- 6-31			− − −	− − −		− − −		− − −

*Transferred to negative herd.

TABLE VIII—*Cows under observation for two years after becoming positive.*

Cow		534	667	992	991	994	562	635	575	570	646	681	424	618	995	850	993
ABORTED		4–29 1926	12–9 1926	8–26 1925	9–11 1926	6–26 1926				7–22 1926	12–27 1928	12–6 1929					
TEST	DATE	REACTIONS															
1	3–25–27	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ − −	+ + +	+ + +	+ ± −	− − −	− − −	+ + +	− − −	− − −	− − −
2	5–25–27	+ + +	+ + +	+ + +	+ + +	+ + ±	+ + +	+ ± −	+ + +	+ + +	+ + −	− − −	+ − −	+ + −	+ − −	+ − −	− − −
3	7–25–27	+ + ±	+ + ±	+ + +	+ + ±	+ + ±	+ + +	+ ± −	+ + −	+ + −	+ + ±	− − −	+ − −	+ + ±	+ ± −	+ + ±	+ ± −
4	9–24–27	+ + +	+ + ±	+ + +	+ + −	+ ± −	+ ± −	+ + −	+ + +	+ − −	+ − −	− − −	+ − −	+ − −	+ − −	+ − −	+ − −
5	12–10–27	+ + ±	+ + +	+ + +	+ − −	+ − −	+ + +	+ ± −	+ ± −	+ − −	+ ± −	− − −	+ − −	± − −	+ − −	+ − −	+ − −
6	2–11–28	+ + +	+ ± −	+ + +	+ − −	± − −	+ + +	+ − −	+ − −	± − −	+ − −	− − −	± − −	− − −	+ − −	± − −	+ − −
7	4– 7–28	+ + ±	+ ± −	+ + +	+ − −	± − −	+ + ±	+ ± −	+ + +	+ − −	+ − −	− − −	± − −	± − −	± − −	− − −	+ − −
8	6–15–28	+ + +	+ ± −	+ + +	+ + −	+ − −	+ + −	± − −	+ + +	+ − −	+ − −	+ + −	+ − −	− − −	− − −	− − −	+ − −
9	8–18–28	+ + +	+ + −	+ + +	+ − −	+ ± −	+ + +	+ + −	+ + +	+ − −	+ ± −	+ + ±	± − −	− − −	− − −	− − −	± − −
10	10–27–28	+ + +	+ + +	+ + +	+ ± −	± − −	+ + +	+ + +	+ + +	+ − −	± − −	+ + +	+ ± −	− − −	− − *	* − −	− − −
11	1– 5–29	+ + +	+ − −	+ + +	± − −	± − −	+ + +	+ − −	+ + −	± − −	+ + +	+ + ±	± − −	− − −	− − −	− − −	− − −
12	3– 9–29	+ + +	± − −	+ + +	± − −	± − −	+ + +	± − −	+ + +	± − −	+ − −	+ + +	± − −	− − *	− − −	− − −	− − *
13	4–19–29	+ + ±	± − −	+ + +	± − −	+ − −	+ + +	+ − −	+ + ±	± − −	+ − −	+ + +	− − −	− − −	− − −	− − −	− − −
14	6– 4–29		− − −	+ + +	± − −	± − −	+ + +	+ ± −	+ + ±	− − −	+ ± −	+ + +	− − −	− − −	− − −	− − −	− − −
15	7– 9–29		± − −	+ + +	± − −	± − −	+ + +		+ + +	± − −	+ + ±	+ + +	− − *	− − −	− − −	− − −	− − −
16	8–10–29		+ − −	+ + +	± − −	+ ± −	+ + +	+ + ±	+ + +	± − −	+ + +	+ + +	− − −	− − −	− − −	− − −	− − −
17	9–14–29		− − −	+ + +	− − −	± − −	+ + +	+ + −	+ + +	± − −	+ + ±	+ + +	− − −	− − −	− − −	− − −	− − −
18	10–19–29		− − −	+ + +	− − −	± − −	+ + +			− − −				− − −	− − −	− − −	− − −
19	12–21–29		+ + −	+ + +	− − −	+ − −	+ + +		+ + +	− − −	+ + +	+ + +		− − −	− − −	− − −	− − −
20	3–29–30		+ + +		− − *	− − −				+ + −		+ + +		− − −	− − −	− − −	− − −
21	4–29–30				− − −	± − −				± − −		+ + +		− − −	− − −	− − −	− − −
23†	7–31–30				− − −	− − −						+ + +		− − −	− − −	− − −	− − −

*Negative. Transferred to negative herd.
†In tests 22 (5-31-30) and 24 (11-19-30), cows 991, 618, 995, 850 and 993 were negative. In test 25 (8-6-31), cows 995, 850 and 993 were negative.

dilutions. Only one of the whole group (cow 991) had ever aborted. None ever again became positive and there is no reason to believe that any of them were actually infected at the time they were placed with the clean animals. Great care should be used in returning animals from the positive herd, as is illustrated in table III, with cows 667 and 570. Both of these passed two clean tests and then again became reactors. Our practice was to isolate the animal after she had passed two clean tests with a 60-day interval. Then, at the next test, if she were still negative, she might be returned to the negative herd. Even this may not be safe in all cases as it is well known that pregnant animals may fail to react for a time prior to abortion.

Cows Under Observation for Two Years After Becoming Positive

Table VII is introduced for the purpose of showing how the reaction is sustained over a long period of time. It necessarily includes animals found in other tables. Hadley and Welsh,[2] working with 26 artificially infected animals, found that only five retained any degree of reaction at the end of two years. The practical question at issue is whether a sufficient number of positive animals will become negative in any reasonable time so that they can be safely returned to the clean herd and the procdeure economically justified.

Sixteen positive cows were under observation for two years or more. Eight either had aborted or did so during the period. Of these, only one became negative for a sufficient time to be returned to the herd. It was three and one-half years after the abortion before she was returned. One and one-half years later, she was slaughtered because she had become unprofitable. Of the eight that did not abort, three persistently reacted for two years or over and five were returned to the clean herd. It will be observed that only one of the five was ever a complete reactor at the 1:200 dilution and she on only one test. Therefore they may be said to be low reactors. We may say with Hadley and Welsh that such animals do tend to lose their agglutinins. The average isolation period of these low reactors was approximately one year and nine months. Since most authors agree that reactors are less profitable than non-reactors, there is a grave question whether retention of even low reactors in the hope of returning them to the herd is a practical procedure.

Conclusions

In the control of abortion disease by means of the blood-test and segregation, the dangerous animal is the infected cow that

aborts in the clean herd before becoming a positive reactor. During the eradication process it is unsafe to return an aborter to the clean herd in less than one year after her isolation. After the herd is clean, these negative aborters may be disregarded except for a 30-day isolation period. While a few positive animals, especially the low reactors, will become negative and can be safely returned to the herd, the percentage is small and the procedure is of questionable value. Except in special cases the double-herd system is a failure and in most instances the immediate elimination of all reactors and aborters will be found most economical.

REFERENCES

[1]Colo. Agr. Exp. Sta. Bul. 317.
[2]Hadley, F. B., and Welsh, W. E.: On the persistency of the agglutination reaction for *Brucella abortus*. Corn. Vet., xxi (1931), 3, pp. 286-291.

Cuba To Be Represented

Captain Rafael Santa Maria, president of the National Veterinary Association of Cuba, will represent that organization at the Atlanta convention, and will press the invitation for the A. V. M. A. to hold a convention in Havana. Those who attended the meetings in Los Angeles (1930) and Kansas City (1931) will recall the warm invitations extended on those occasions by Dr. Clodoaldo A. Arias, of Havana. Efforts are being put forth to organize a post-convention tour of the Pearl of the Antilles. A special round-trip rate of $50.32 from Atlanta to Havana has been authorized by the Central of Georgia Railways in coöperation with the P. & O. Steamship Co. Those who take advantage of this offer have the privilege of going by way of Key West and returning by way of Tampa, or vice versa. The Hotel Plaza, Havana, has arranged special low rates for the occasion: $2.50 and up for single rooms, $2.00 per person for double rooms.

Parking Space

Free parking space for automobiles will be available near the Atlanta Biltmore Hotel. For those who wish to store their cars during the convention, ample and convenient storage will be found near the hotel.

Southern Railway Veterinarians

Veterinarians connected with the Southern Railway will hold a meeting Thursday afternoon, August 25, immediately following the adjournment of the general session. The meeting will be held in Room 10, on the Mezzanine floor.

ANEMIA AND ERYTHROLEUCOSIS OCCURRING SPONTANEOUSLY IN THE COMMON FOWL*

By E. L. STUBBS *and* J. FURTH

The Henry Phipps Institute and the Veterinary School, University of Pennsylvania, Philadelphia, Pa.

Erythroleucosis is a transmissible disease of the common fowl characterized by severe anemia with numerous lymphoid cells, presumably precursors of erythrocytes, in the peripheral circulation, and by a selective accumulation of these lymphoid cells in the bone-marrow and spleen, and in the capillaries of many other organs. Ellermann,[1] the first to observe this condition, thought that erythroleucosis is caused by a filtrable virus that also gives rise to myeloid and to lymphoid leucosis† and to simple anemia.

Ellermann's view that erythroleucosis is a distinct disease has recently been confirmed.[2] It has been observed also that erythroleucosis and myeloid leucosis occur among passages of a single transmissible strain, but no evidence was found to support the view of Ellermann that lymphoid leucosis is caused by the same agent that transmits the other two.[3,4]

Our knowledge of erythroleucosis is derived from observations of this disease as produced by transmission. There are no reports, as far as we are aware, of instances occurring through natural means. In England, Bedson and Knight[5] described a severe spontaneous anemia of the fowl characterized by a great increase of lipochrome, in the skin, beak, leg-scales, eye-ring, blood-plasma and body fat. Blood of the anemic fowls injected into healthy hens produced either transient anemia or polycythemia. Bedson and Knight were inclined to consider this anemia different from the conditions observed by Ellermann. Cases such as those observed by Bedson and Knight were reported by Bayon,[6,7,8] who has suggested the term "erythromyelosis." Bayon found in the small intestine in most, but not in all, of the anemic fowls, a minute cestode, *Davainea proglottina*, which he thought produced a predisposition to the attack of an intracellular virus (microplasm). The results of his transmission experiments were similar to those of Bedson and Knight. Only one among many fowls to which he attempted to transfer this disease developed a progressive anemia. Bedson and Knight,

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†Leucosis is used as a common term for all types of leucemias and related conditions. According to the type of cell involved it is divided into lymphoid, myeloid and erythroleucosis (Ellermann).

and Bayon described the anatomical changes of organs observed in their spontaneous cases, but stasis of lymphoid cells, the most striking feature of erythroleucosis,[2] is not mentioned by them.

Thus there are no descriptions of erythroleucosis as a disease occurring among fowls in their natural environment. Among fifty cases of leucosis in flocks in Delaware, Dr. Frank Hare observed, in coöperation with us, three that on microscopic examination resembled erythroleucosis. Moreover, Dr. W. H. Feldman, of Minnesota, sent us sections of organs of an anemic fowl that showed the characteristic appearance of transmissible erythroleucosis. Apparently it does occur in this country, but its symptoms are not well known. Dr. R. O. Biltz, becoming familiar with the disease produced by transmission, called our attention to the "endemic" occurrence of this condition on a poultry farm in Pennsylvania. Through his courtesy and that of of Dr. R. L. Elsea, we obtained nine fowls from this farm. Two of these had lymphoid leucosis and one a large round-cell sarcoma; five had severe anemia resembling erythroleucosis. A description of the latter cases is the subject of this report.

Histories of Cases of Spontaneous Anemia Resembling Erythroleucosis

The fowls described here were hatched in February, 1930. In the year that followed, most of this flock died, according to the owner, with symptoms of paralysis, tuberculosis, big liver, big spleen, or anemia. Two of the fowls to be described were received February 18, 1931; the rest, March 11, 1931. All were Barred Plymouth Rock hens. Data in table I are given as introduction to the description of these cases; detailed descriptions will follow.

Table I—*Data on five spontaneous cases.*

Fowl	Blood Picture	Postmortem: Erythroleucotic Involvement	Attempts at Transfer
1	Severe anemia	Very slight	Successful
2	Severe anemia	None	Negative
3	Severe anemia	None	Successful
4	Erythroleucosis	Severe	Successful
5	Severe anemia	None	Transfer not attempted

Fowl 1: In good condition when received, except that the comb appeared pale. In blood smears (table II) abundant polychrome erythrocytes, polychrome erythroblasts, and a few basophile erythroblasts were seen. Following the removal of blood for the purpose of transfer, the chicken was killed.

The breast muscle was well developed. There was abundant yellowish fat in the abdominal cavity. The weight of the liver was sixty grams. It was distinctly yellow in color, and appeared to be fatty and spotted with several minute reddish areas. The spleen

weighed four and one-half grams and was reddish, with prominent greyish follicular structures. The mucous membrane of the duodenum and of the distal end of the ceca was thickly studded with minute hemorrhages. In other parts of the intestine, hemorrhages were less numerous. On gross examination no parasites were found and smears showed no coccidia or Davainea. The heart muscle was pale and the pericardium was spotted with petechial hemorrhages. The marrow cavity of the femur and of the tibia was almost entirely obliterated by spongy bone (fig. 7) but centrally there was some firm grayish-white, marrow-like substance. The bones were very hard.

On microscopic examination of the liver, an extensive accumulation of small round cells was seen in the portal area. In a very few areas there was stasis of lymphoid cells, characteristic of erythroleucosis. This could easily have been overlooked, for the bulk of the liver did not show this change. The blood clot of the larger vessels showed the picture of anemia with many lymphoid cells (fig. 10). The liver cells were filled with large vacuoles, presumably fat. There was mild stasis of lymphoid cells in the pulp of the spleen, but the normal lymphocytic elements showed no marked abnormalities.

The cortex of the femur showed vacuolization, some places being thickened, some thinned. The cavity of the marrow was filled with spongy bone, the spaces of which were filled by fibrous bands and with cells the majority of which resembled granulocytes. The tissue was not preserved sufficiently for all cells occupying the marrow to be recognized with certainty, but cells resembling erythroblasts seemed scant.

The intravenous inoculation of 0.5 to 4.0 cc of blood of this fowl into five Barred Plymouth Rock fowls about four months of age caused leucosis in three of them; two recovered. (See table III.)

Fowl 2: The general appearance and blood-picture of this hen (fig. 2) was similar to the one just described. The white-blood-cell count was approximately 33,000; the red-blood-cell count, 1,365,000, and the figure for hemoglobin was 28 (Sahli). This fowl was observed during a period of four months, but there was no noteworthy change in the composition of the peripheral blood. It was killed after the removal of 15 cc of blood for purposes of transfer. The body weighed 2100 grams and was well nourished. In the abdomen there was a large amount of very yellowish fat. The liver weighed 80 grams and was brownish red in color. It was mottled throughout with many minute pale areas. The spleen weighed 3 grams, was reddish brown in color and appeared normal. The duodenum showed many small hemorrhages in the serous coat. The mucous membrane of the duodenum was swollen and brown in color. No parasites could be detected on gross examination and in smears no Davainea or coccidia were found. The ovary was inactive and contained degenerated ova, which were brownish in color.

The long bones were very hard. The bone-marrow cavity was almost obliterated by a honeycombed, bone-like substance, containing in some places small amounts of rather firm, pale-yellowish, marrow-like material. The bones of the extremities as well as the breast bone were difficult to break. The wing bones contained air spaces.

On microscopic examination, the liver (fig. 9) showed extensive infiltration with granulocytic tissue in the portal area consisting chiefly of myelocytes, small scattered accumulations of lymphocytes, chiefly in the portal area, and in the Kupfer cells, yellowish pigment and cell débris. The blood clot seemed anemic but there was no stasis of lymphoid cells in the capillaries. The bone-marrow contained no fat; it was hyperplastic, but the hyperplasia was

unlike that seen in transmissible leucosis. It was formed chiefly by small round cells resembling lymphocytes and areas resembling hypertrophied germinal centers. There were scattered polymorphonuclear leucocytes and myelocytes. The monoculear phagocytes of the spleen contained abundant yellowish pigment; there was some lymphoid hyperplasia but no sign of erythroleucosis.

Injections of 5 cc of blood into each of three Barred Plymouth Rock fowls four months old were without effect.

Fowl 3: The general appearance of the third hen was similar to the two fowls described above. From the date of receipt (March 13) until its death (May 12), the blood-smears of this fowl exhibited the picture of severe anemia. The morphological changes in the blood will be described in table II and in the text following.

At postmortem examination, this fowl, in contrast to fowls 1 and 2, was much emaciated. The organs were very pale. The liver was slightly enlarged, yellowish brown and spotted with many pale grayish and yellowish areas, varying in size from that of a pinpoint to about 2 mm. The spleen was pale grayish brown and appeared to be of normal size. The pericardium contained much yellowish, gelatinous material. The thymus appeared small and atrophied. The cavity of the long bones was narrowed by spongy bone (fig. 6) and the marrow was pale yellowish and gelatinous (fig. 4).

On microscopic examination no organ showed stasis of lymphoid cells. The blood-clot indicated severe anemia. The monoculear phagocytes of the liver and spleen contained much yellowish pigment. The liver cells also were filled with similar but finer pigment spots. The bone-marrow (fig. 4) contained much homogeneous gelatinous material and showed slight granulocytic hyperplasia.

Two of the five young chickens inoculated with blood of this fowl died of erythroleucosis 67 and 54 days after inoculation.

Fowl 4: Erythroleucosis developed in this Barred Plymouth Rock hen while under observation in the laboratory and the fowl died in four weeks. The fowl was in good condition when received (March 11) and the blood-smear appeared normal. Ten days later, the blood (fig. 1) showed the characteristic appearances of erythroleucosis (table II) and subsequent smears indicated rapid progression of the disease. On March 23, mitotic figures were already seen in the blood, but death did not occur until April 22.

The carcass weighed 1510 grams and showed no sign of emaciation. The liver was much enlarged (estimated weight, 100 grams), grayish brown and spotted thickly and uniformly with just visible, minute, yellowish, gray or reddish spots. The spleen was grayish brown and very much enlarged (estimated weight, 15 grams). The mucosa of the duodenum was thickly studded with minute hemorrhages. In the small intestine, including the duodenum, there were several round worms and many tapeworms. In smears no Davainea were seen. The pericardium contained considerable amounts of clear serous fluid and the heart muscle was pale brown and spotted on its pericardial surface with a few petechial hemorrhages. The thymus was small. The bone-marrow was pale, grayish brown, and of increased consistency. The obliterating spongy-bone formation, seen in the first three fowls described, was not found in the marrow of this fowl.

The microscopic appearances of the organs were indistinguishable from erythroleucosis produced by transmission. There was marked stasis of lymphoid cells in the liver (fig. 8) and spleen; the hyperplastic marrow was almost entirely composed of such cells (fig. 5).

Blood taken from the heart, about one to two hours after death, caused erythroleucosis in one of three young fowls injected intravenously.

Fowl 5: The history of this hen is similar to that of the first three cases described. The blood-picture indicated severe anemia (table II). Only once (March 16) during the period of observation (March 11 to June 24) was a mitotic figure seen in the blood. At postmortem the fowl weighed 1700 grams, the liver 64 grams, and the spleen 1 gram. There was a large amount of very yellowish fat in the abdomen; the liver was likewise yellowish with a faint reddish mottling. The duodenum was reddish brown, the mucous membrane ridged. The mesentery was thickened and reddened. The ovary was inactive and contained several brownish degenerated ova. The long bones were difficult to break; the marrow cavity was almost entirely obliterated by the formation of spongy bone, but there were traces of grayish bone-marrow in some parts.

On microscopic examination the liver was found to be thickly spotted with necrotic areas. There were many nodular accumulations of large cells like lymphocytes in the portal area. The Kupffer cells contained abundant yellowish pigment. Anemia was not marked in the blood-vessels and stasis of lymphoid cells was not seen. There was abundant phagocytosis of pigment particles in the spleen and still more in the duodenum. This organ was thickly surrounded with large monoculear cells. No organ showed signs of erythroleucosis.

Relation of Spontaneous Anemia to Erythroleucosis

The five cases of spontaneous anemia are not uniform in character. Four resemble the severe spontaneous anemia adequately described by Bodson and Knight, and Bayon; and only one (fowl 4) resembled erythroleucosis as produced by transmission. On injection of blood of the anemic fowls into healthy fowls, several of the inoculated birds developed erythroleucosis and none simple anemia. It seems that both are caused by the same transmissible agent, and spontaneous development apparently favors the type of anemia described by the investigators named above, whereas artificial transmission favors erythroleucosis. Transitional forms likewise seem to occur (fowl 1). In the following, an attempt will be made to compare these two types of anemia and their intermediate forms.

Clinical Picture

In severe chronic anemia there is marked yellowish discoloration of the skin and most of its appendages. In erythroleucosis there is some paleness, but in the early stages the fowl may seem to be healthy, while the blood-picture may show pronounced erythroleucosis. Loss of egg-production and emaciation follow as the disease progresses.

Spontaneous anemia, when caused by a transmissible agent, seems to be chronic. The cases observed by us were pronounced when the fowls were received, yet one of them lived two and one-

third months, and another, not showing any progression, was killed after an observation period of three and one-half months. Similar cases observed previously among the fowls inoculated with leucemic material seem to follow a like course.[2] Erythroleucosis is usually acute or subacute.

THE BLOOD-PICTURE

The blood-picture in chronic anemia (fig. 2) shows numerous polychrome erythrocytes and polychrome erythroblasts but few basophile erythroblasts. Accurate criteria for differentiating the type of anemia described by Bedson and Knight from ordinary anemia as readily produced by hemotoxic substances are wanting, but in erythroleucosis (fig. 1) the basophile erythroblasts are relatively numerous.[2] This is well supported by the differential counts of the five spontaneous cases (table II).

NOTES ON THE BLOOD CHANGES DESCRIBED IN TABLE II

The differential counts given are only approximate, because the classification of some cells is arbitrary. The cells seen in erythroleucosis were described in a previous report[2]. Basophile erythroblasts (erythroblasts that seem to be entirely free from hemoglobin) were not counted among the white corpuscles.

Fowl 1: There was some leucopenia and agranulocytosis. Thrombocytes seemed absent. The lymphoid cells with vacuoles did not seem to be primitive. The white-blood count was 57,500, the red-blood count 1,155,000, and hemoglobin 23 (Sahli).

Fowl 2: In smear of February 18, one mitotic figure was seen. The white-blood count was 33,500, the red-blood count 1,365,000, and hemoglobin 23 (Sahli).

Fowl 3: The differential counts of the last smear of this fowl, taken May 9, have not been included because most of the leucocytes seem atypical. The majority of the cells (about 42 percent) resembled large mononuclears, many being perhaps primitive. Many of them contained globular inclusions of various sizes (fig. 3); the larger ones had a dark-stained, ring-shaped outline with a faintly pink-stained central part. Aside from these inclusions many cells contained fine purple granules, with or without the globular inclusions. There were also free cytoplasmic masses, some of them containing the type of inclusion just described. About 16 per cent of the cells were small lymphocytes and about 10 per cent medium-sized, apparently primitive, lymphoid cells. Ordinary polymorphonuclears were absent. Many of the lymphoid cells or small cells resembling lymphocytes had vacuoles in the cytoplasm, probably atypical thrombocytes. Among 100 leucocytes, two basophile erythroblasts were seen and one leucocyte was in mitotic division. Polychrome erythrocytes and erythroblasts were abundant.

Fowl 4: A smear of this fowl, taken April 21, resembled the smear of March 28.

Only in fowl 4 (erythroleucosis) was the number of basophile erythroblasts considerable (32 per cent). In fowl 1, with slight erythroleucotic involvement, there were 16 basophile erythroblasts per 100 leucocytes and in none of the three animals with anemia

TABLE II—*Blood changes in spontaneous anemia and erythroleucosis.*

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FOWL	DATE OF EXAMINATION	WHITE CELLS (ESTIMATED)	POLYCHROME ERYTHROCYTES	POLYCHROME ERYTHROBLASTS	BASOPHILE ERYTHROBLASTS PER 100 WHITE CELLS	DIFFERENTIAL COUNTS							
						POLYMORPHONUCLEAR LEUCOCYTES*	MONOCYTES	LARGE MONONUCLEAR CELLS WITH PURPLE GRANULES	MAST CELLS	LYMPHOCYTES	LARGE LYMPHOCYTES (PRIMITIVE)	LYMPHOID CELLS WITH VACUOLES	MITOTIC FIGURES
1	2–19–31	Slight decrease	Abundant	Abundant	23.5	0.5	3	—	1.5	76.5	3	15	0.5
2	2–18–31	Normal	Abundant	Abundant	3.5	16.5	2.5	1	3	72.5	4		0.5
	3–11–31	Normal	Abundant	Abundant	1	22	12	3.5	1.5	59.5	1	0.5	—
	6–26–31	Normal	Few	Few	1	27	10	3	1	59			
3	3–16–31	Slight increase	Many	Many	—	16.5	2.5	—	—	79.5	0.5	1	—
	3–28–31	Slight increase	Abundant	Abundant	0.5	7	9	—	0.5	79.5	2.5	1.5	—
4	3–13–31	Moderate increase	None	None	—	58	30	—	0.5	11.5	—	—	—
	3–16–31	Moderate increase	Very occasional	None	—	48.5	18.5	—	0.5	33.5	—	—	—
	3–23–31	Slight increase	Few	Few	5.5	52	12.5	1	5	27	2	—	0.5
	3–28–31	Slight increase	Abundant	Abundant	36	39	15.5†	—	2.5	33.5	5.5	1	3
5	3–16–31	Slight increase	Many	Many	3	10	8	0.5	1	80	0.5	—	—
	4–21–31	Slight increase	Few	Occasional	0.5	0.5	7	0.5	1	88	3	—	—
	5– 8–31	Normal	Many	Few	1.5	22	8.5	—	0.5	67	2	—	—

*Including a few myelocytes.

†Seven large mononuclear cells appeared primitive.

were there more than five basophile erythroblasts in 100 leucocytes.

Associated with the changes in the erythropoiesis there seems to be a marked disturbance in granulocytopoiesis. Polymorphonuclear leucocytes were absent in the blood of fowl 1 and in the terminal stage in fowl 4.

The presence of large, apparently primitive mononuclear cells, presumably myeloblasts, indicates myeloid involvement, as often seen in association with erythroleucosis produced by transmission.

The formation of thrombocytes seems likewise profoundly disturbed. Normal forms are practically absent. Many cells like thrombocytes have large vacuoles in the cytoplasm.

Annular cell inclusions not unlike those seen in the blood of fowl 3 were found by Macfie,[9] in Nigeria, and by Adler,[10] in Palestine, in the mononuclear leucocytes of fowls suffering from an acute infectious disease. One of the animals described by Adler had leucemia, but none of them had pronounced anemia.

Anatomical Changes

In both severe chronic anemia and erythroleucosis the comb and wattles and unfeathered parts of the skin were pale and sometimes distinctly yellowish. In advanced cases there is emaciation with a wasting away of the breast muscles. In severe chronic anemia the fat of the abdominal cavity is often present in considerable amount, and is very yellowish. In both conditions hemorrhagic spots are found in many parts of the carcass such as the muscles of the breast and of the femoral region, the pericardium and the peritoneum.

The spleen in severe chronic anemia may be of normal size, or it may be slightly enlarged or slightly decreased in size. In the fowls described here, this organ weighed 1 to 3 grams. The weight of the spleen in 24 instances of anemia described by Bayon varied between 0.5 and 7 grams, with an average weight of 1.8 grams. In erythroleucosis the spleen is much enlarged. The organ weighed 15 grams in the spontaneous case described here. In fowl 1, with an intermediate form of the disease, it weighed 4.2 grams. The small spleens are pale brown; the enlarged spleens, reddish or greyish brown. Very much enlarged spleen, with an only slightly enlarged liver, is one of the most characteristic observations in erythroleucosis.

The bone-marrow, in the instances of severe chronic anemia studied, was replaced almost entirely by a formation of spongy bone proliferating from the endosteum (figs. 6 and 7). The changes in the bones resemble those with osteitis fibrosa. The character of the free marrow is variable.

The liver in these cases of anemia did not seem to show any characteristic changes on gross examination. It was not enlarged or only slightly enlarged; its color was brown, often yellowish brown. In three of our cases the weight of the liver was 60, 64 and 80 grams, and the weight of the liver in the cases described by Bayon varied between 15 to 104 grams, with an average of 43 grams. The liver in erythroleucosis is slightly or moderately enlarged, reddish brown or grayish brown, often showing a fine mottling caused by pale grayish areas.

The intestines are usually the site of mild, hemorrhagic enteritis. The mucous membrane is red or reddish brown and the walls are thickened.

The microscopic changes in the five instances of anemia that are described may be briefly stated as follows: In fowl 4 they were characteristic for erythroleucosis. The bone-marrow (fig. 5) showed extensive hyperplasia of lymphoid cells and there was stasis of these cells in the liver (fig. 8) and spleen. The three instances of anemia (fowls 3, 2 and 5) were characterized by signs of blood destruction such as were evidenced in the liver and spleen and in the bone-marrow by numerous cells laden with yellowish pigment giving the iron reaction with ferrocyanid of potassium. In all these cases there was a formation of osteoid tissue replacing much blood-forming marrow (figs. 6 and 7) and lack of hyperplasia of erythroblastic elements. The case of fowl 2, which is intermediate, shows a few areas in the liver with pronounced stasis, the bone changes mentioned above, and lack of increased erythrogenesis in the marrow, with little evidence of blood destruction.

Transmission Experiments

Technic: Blood was drawn from the wing vein into a chilled syringe containing heparin solution (1:1000), one-tenth in volume that of the desired amount of blood. The blood was immediately injected intravenously into young birds in doses varying from 0.5 to 4 cc.

Transmission of the anemia of the four fowls described here was attempted, in three of them with success. In table III, details of these transmissions are given:

The results of the transmissions given in table III show the intimate relation between severe chronic anemia and erythroleucosis. The failure of Bedson and Knight to produce erythroleucosis by intravenous inoculations of blood of the anemic fowls observed by them may be due to the circumstance that transfer was attempted to only a small number of fowls and that these were full-grown birds. Our observations[11] indicate that when inoculated a smaller percentage of older birds will develop the

disease than younger birds and that recovery is more common among the older birds.

Lymphoid leucosis did not occur among these passages.

DISCUSSION

The cases of spontaneous anemia and erythroleucosis described here serve to call attention to some problems connected with the transmissible anemia of the fowl.

Anatomically erythroleucosis is severe anemia in which there is an enormous increase of lymphoid cells, precursors of erythrocytes, in the peripheral circulation and especially in the organs, such as the bone-marrow, liver and spleen. Anatomically there are transitional forms between severe anemia and erythroleucosis, but etiologically certain anemias and erythroleucosis seem intimately related, as was recognized by Ellermann.[1] Most of the spontaneous cases seem to be associated with the picture of severe anemia, like those described by Bedson and Knight, and Bayon, but the disease produced by transmission seems almost exclusively erythroleucosis. This behavior made it difficult to establish the etiological connection between the two. Criteria to differentiate severe anemia transmissible by a filtrable agent from ordinary anemias are not known.

TABLE III—*Transmissions of spontaneous anemia and spontaneous erythroleucosis.*

DONOR	FOWLS INOCULATED	SUCCESSFUL INOCULATIONS	UNSUCCESSFUL INOCULATIONS	SUCCESSFUL INOCULATIONS		
				PERIODS OF INCUBATION* (DAYS)	DURATION OF SICKNESS (DAYS)	BLOOD-SMEAR
1	5	3	2	28 28 65	11 29 14	Erythroleucosis Erythroleucosis Erythroleucosis
2	3	0	3	—	—	—
3	5	2	3†	45 41	22 13	Erythroleucosis Erythroleucosis
4	6	1	5	21	80	Erythroleucosis

*Number of days between inoculation and appearance of changes in the circulating blood.

†One of these died from an intercurrent infection; the blood-smear before death and the microscopic appearance of the marrow suggested incipient erythroleucosis.

The objection might be raised that in the transmission experiments reported here the test animals were kept in one room, with fowls afflicted with erythroleucosis produced by a trans-

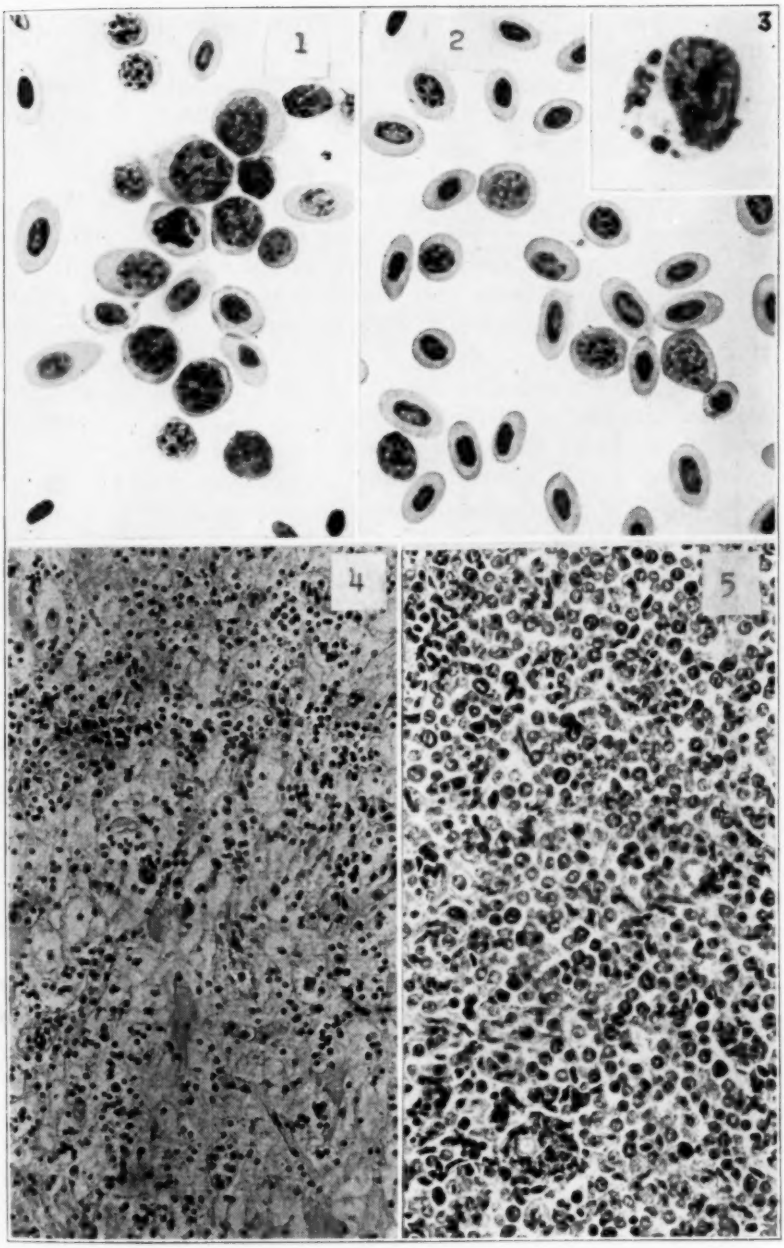

PLATE I

missible agent of great virulence. But previous experiments[3,4] showed that the chance of spontaneous transfer under the conditions of our laboratory is remote.

Some of the properties of the transmissible agent of erythroleucosis are as follows: It passes Berkefeld filters that retain small visible microbes. It resists repeated freezing and thawing and treatment with distilled water. It becomes inactive at 37° C. for a few days, but retains its activity at 4° C. for at least seven days. It resists drying and can be preserved in glycerin.[12] Much of the active agent in the blood is associated with the cells, but cell-free plasma also transmits the disease.[13] It affects all breeds of the domestic fowl, but other birds resist inoculation.[11]

These properties suggest that the filtrable agent of erythroleucosis may be an ultramiscropic virus. The high concentration of the agent in the blood points perhaps to the possibility that blood-sucking insects may act as vectors. Experiments to determine the possibility of spontaneous transfer are few and inconclusive. That blood-sucking insects may be vectors of transmissible leucemia has been considered but not proved.[14, 15] Bayon[6, 8, 7] assumes that accessory factors such as tapeworm infestation activate an intracellular virus, called by him "microplasm," but his suggestion, likewise, lacks experimental proof.

SUMMARY

Three instances of severe chronic spontaneous anemia, one spontaneous erythroleucosis and one that appeared intermediate, were observed in a flock of fowls in Pennsylvania.

Pallor with a progressive disturbance of health were the most conspicuous symptoms. Simple anemia follows a more or less chronic course, whereas erythroleucosis seems to be relatively acute but sometimes of considerable duration. The blood-smears in erythroleucosis in contrast to simple anemia show relatively large numbers of very young basophile erythroblasts.

The gross postmortem changes were paleness of the internal organs, in some cases an intense yellowish coloration of the body fat, skin and most of its appendages, emaciation, petechial and ecchymotic hemorrhages. The spleen is only slightly enlarged. In most cases of anemia the bone-marrow is replaced by spongy bone formation with no hyperplasia of marrow, whereas in erythroleucosis there is extensive accumulation of basophile erythroblasts which show little or no tendency to undergo maturation. The liver in anemia may show little change but in erythroleucosis it is moderately enlarged and reddish brown or grayish brown in color.

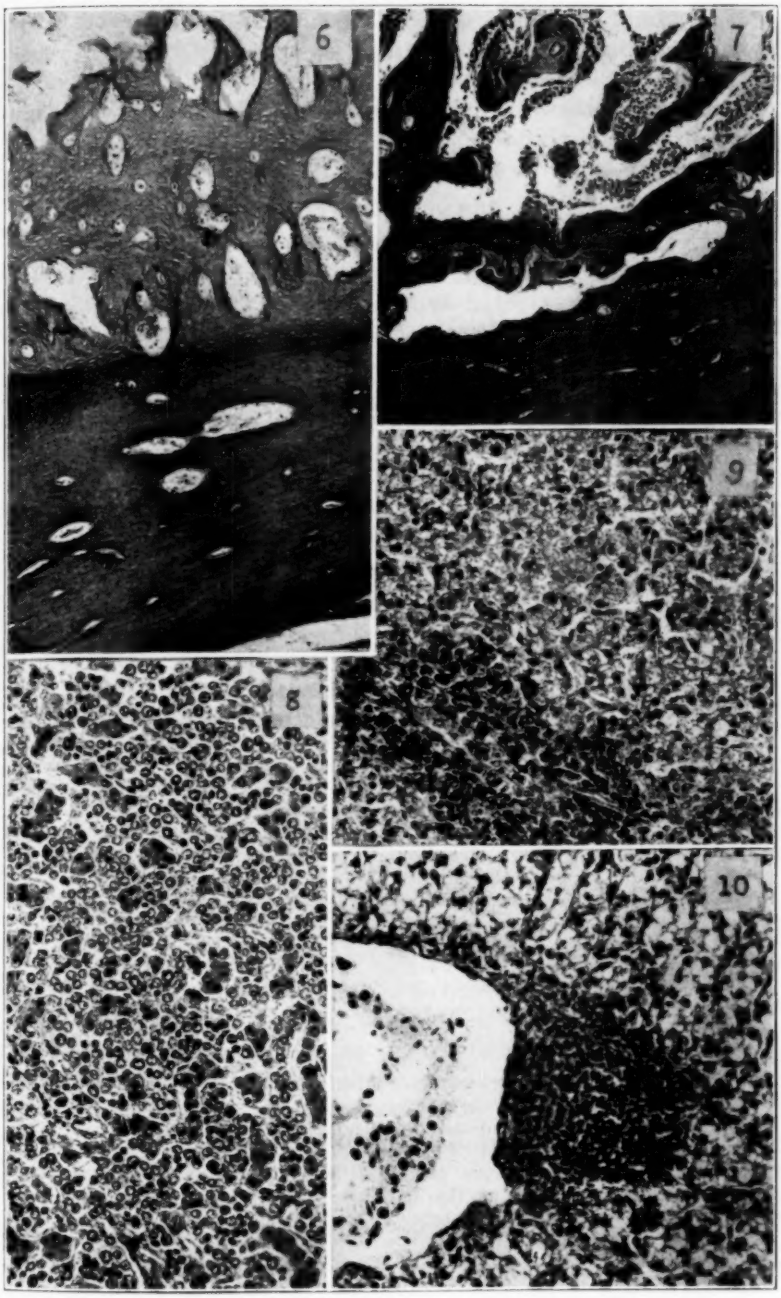

PLATE II

Transmission experiments with three of four spontaneous cases were successful. Only one of these spontaneous cases was well-defined erythroleucosis, whereas the disease produced by transmission was either erythroleucosis or myeloid leucosis. This observation shows the close relationship between the anemia observed and erythroleucosis.

ACKNOWLEDGMENT

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DESCRIPTIONS OF FIGURES

PLATE I

FIG. 1. A blood-smear from fowl 4 with spontaneous erythroleucosis. Note the large number of young erythroblasts in proportion to mature erythrocytes. In the central part of the field there is a mitotic figure (x 900).

FIG. 2. A blood-smear from fowl 2 with severe chronic anemia. The number of erythroblasts in proportion to erythrocytes is smaller than in erythroleucosis and there are no very young forms of erythroblasts (x 900).

FIG. 3. Inclusion bodies in the cytoplasm of a large mononuclear leucocyte of fowl 3 with severe chronic anemia (x 900).

FIG. 4. The bone-marrow of fowl 3 with severe chronic anemia; no erythroblastic hyperplasia (x 180).

FIG. 5. The bone-marrow of fowl 4 with spontaneous erythroleucosis; almost the entire marrow occupied by primitive (basophile) erythroblasts (x 200).

PLATE II

FIG. 6. Thickening of the wall of the femur (fowl 3) and formation of massive osteoid tissue narrowing the marrow cavity (x 180).

FIG. 7. Spongy bone and osteoid tissue (fowl 1) proliferating from the endosteum occupy most of the marrow cavity (x 180).

FIG. 8. Stasis of primitive (basophile) erythroblasts in the liver of fowl 4 with spontaneous erythroleucosis (x 180).

FIG. 9. Granulocytic and lymphocytic hyperplasia in the portal area of the liver of fowl 2 with severe chronic anemia. There is no stasis of lymphoid cells in the capillaries of the liver (x 180).

FIG. 10. The liver of fowl 1 with severe chronic anemia, showing a large sinusoid with numerous erythroblasts but no stasis in the capillaries. There is a nodular accumulation of small lymphocytes adjacent to the sinusoid (x 180).

OBSERVATIONS ON THE LONGEVITY OF MYCOBACTERIUM TUBERCULOSIS OF BOVINE ORIGIN*

By WILLIAM H. FELDMAN, *Rochester, Minnesota*
Division of Experimental Surgery and Pathology
The Mayo Foundation

In the course of a series of experiments pertaining to the suitability of different culture media for the bovine form of *Mycobacterium tuberculosis*, it became necessary to secure a considerable number of tuberculous lesions from cattle infected spontaneously. The animals had all reacted positively to tuberculin and were slaughtered. The material consisted of 21 lymph-nodes, and a piece of tissue from the lung.

The material was packed in dry borax and forwarded to the laboratory. After smears had been made for microscopic examination, approximately 10 gm of the necrotic débris was emulsified in 10 cc of a solution of sterile physiologic sodium chlorid and the heavier particles were permitted to settle out. Two portions of 1 cc of supernatant fluid were used for culture and two portions of 1 cc were used to inoculate two guinea pigs, after which the respective tubes containing the balance of the emulsified lesions were stoppered with cotton and capped with tight-fitting, rubber finger-cots. The tubes containing the emulsions were then placed in a unit of a central refrigerating system in which temperatures between 5 and 10° C. were maintained. The tuberculous lesions were received over a period of five months, and the age of the respective emulsions, when submitted to tests for viability, varied from 310 to 451 days.

METHOD OF DETERMINING VIABILITY

Since previous observations[1] indicated that the inoculation of guinea pigs was a more dependable method for demonstrating viable *Mycobact. tuberculosis* in spontaneous infective material than cultural methods, the inoculation method was chosen. Preparations from each emulsion were stained with carbolfuchsin to determine the presence of acid-fast bacillary forms, after which two guinea pigs were inoculated, each with portions of 1 cc of supernatant fluid. One animal was inoculated subcutaneously and one intraperitoneally. They were then placed two in a cage and those that survived were killed for necropsy after the lapse of 65 days. Those dying before that time were ex-

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amined for lesions of tuberculosis. Histologic sections, appropriately stained by a method previously described,[2] were prepared from the lungs, liver and spleen of each guinea pig, regardless of the presence or absence of gross lesions, in order to reduce the possibility of error to a minimum.

TABLE I—*Longevity of Mycobacterium tuberculosis in emulsions of naturally infected lesions from cattle after prolonged refrigeration: Summary of results.**

EMULSION	SMEAR FROM LESION	CULTURE	ORIGINAL INOCULATION OF GUINEA PIGS	IN REFRIGERATOR (DAYS)	SMEAR FROM REFRIGERATED EMULSION	SECOND INOCULATION OF GUINEA PIGS	
						S	I
1	+	+	+	451	+	−	−
2	+	+	+	450	+	−	−
3	+	+	+	450	+	−	+
4	−	+	+	450	+	+	+
5	+	+	+	446	+	−	−
6	−	−	+	437	−	−	−
7	+	+	+	437	+	−	−
8	−	+	+	436	−	−	−
9	+	+	+	435	+	−	−
10	+	+	+	435	+	−	−
11	−	−	+	422	−	+	+
12	−	−	+	420	−	−	−
13	−	−	+	420	−	−	−
14	+	−	+	418	−	−	−
15	+	+	+	345	+	−	+
16	+	+	+	318	+	+	−
17	−	+	+	312	−	+	−
18	+	+	+	312	+	+	+
19	+	+	+	311	+	+	+
20	+	+	+	311	+	+	−
21	+	+	+	310	+	+	+
22	+	+	+	310	+	+	+

*All of the emulsions represented lesions of lymph-nodes with the exception of emulsion 4, which was prepared from a lesion of the lung tissue.
S = subcutaneously.
I = intraperitoneally.

RESULTS

The results obtained in this study are summarized in table I. Although the entire twenty-two emulsions had originally been proved by the inoculation of guinea pigs to possess the bacillus of tuberculosis, in only eleven could the infective agent of tuberculosis be demonstrated by this means after prolonged exposures to refrigeration. Since acid-fast bacillary forms were present in the same number of emulsions at the time injections were given to the second series of guinea pigs as were present when the emulsions were first prepared, the failure of lesions of tuberculosis to develop in animals of the second series inocu-

lated with certain emulsions cannot be explained on the basis of the disappearance of the acid-fast forms from the emulsions. The conclusion must be that when lesions did not follow inoculation of an emulsion that had previously been infective, the provocative agent was either dead or in an extremely diminished state of animation.

Eight of the emulsions were shown to possess viable *Mycobact. tuberculosis* in sufficient numbers to incite definite lesions of tuberculosis after being in the refrigerator for variable periods of from 310 to 345 days. Of the four emulsions that remained in the refrigerator for from 418 to 422 days, only one induced lesions in the test animals, whereas six emulsions that continued in the refrigerator for from 435 to 446 days were all devoid of viable bacilli of tuberculosis as determined by the inoculation of guinea pigs. Four of the emulsions had been in the refrigerator for from 450 to 451 days when injected, and two of these contained viable and pathogenic forms of *Mycobact. tuberculosis* and two did not.

In six instances, lesions of tuberculosis developed in both guinea pigs after injection with the respective emulsions. In five instances, however, tuberculous lesions developed in only one of the two guinea pigs infected; lesions were not demonstrable in the tissues of the other pig although they were examined microscopically. Lesions of tuberculosis were found in nine of the 22 guinea pigs that were inoculated subcutaneously, and tuberculous lesions were found in eight of the 22 animals that were inoculated intraperitoneally.

Mention should be made also of the fact that cultures were attempted from emulsions 16, 17 and 19 after they had been in the refrigerator for a period of 228 days. These attempts to secure culture of acid-fast bacilli failed, yet each of these emulsions was subsequently found to possess viable and virulent forms of *Mycobact. tuberculosis* when guinea pigs were inoculated approximately 90 days later.

Emulsion 1, which failed to infect guinea pigs after having been in the refrigerator 451 days, had previously been shown to possess virulent bacilli of tuberculosis which also could be cultured after having been in the refrigerator 223 days.

Comment

Several observers have reported on the tenacity of the bovine form of *Mycobact. tuberculosis*. Without attempting to review all the work which has been published previously, it would seem opportune to summarize briefly some of the more important facts that have been determined concerning the viability of this microörganism under different conditions of environment.[3]

Smith[4] kept fully developed cultures of the human and bovine forms of the bacillus of tuberculosis at 40 to 50° F. and found that, although the number of viable organisms in such cultures is small, some remained virulent for guinea pigs for from seven to nineteen months. He found that bovine forms of the organism are more resistant than the human form, and that certain strains occur which will multiply in guinea pigs but which will fail to do so on culture mediums.

Corper and Gauss,[5] working with a bovine strain of *Mycobact. tuberculosis* that had been isolated about a year previously, found that when grown on Petroff's gentian-violet-egg medium and held at ice-box temperature, the cultures were viable after six months, whereas those continued at incubator temperature remained viable after sixteen months. Tests for virulence were not made.

A strain of the bovine bacillus of tuberculosis, obtained by Soparkar[6] from the lung of a deer that had died in the Zoölogical Gardens of Bombay, was found to be more resistant to direct sunlight and diffuse sunlight than the bacillus of human origin. When exposed to direct sunlight the bovine form was viable after ten hours, whereas the human form remained viable for only six hours. When exposed to diffuse sunlight the bovine form of the organism remained virulent for as long as 30 days. When exposed to a 32-candle-power electric light, the bovine form was alive after 74 days, but was found to be nonviable after 100 days.

Gloyne[7] also reported on the tenacity of what was probably the bovine form of bacillus of tuberculosis. In July, 1914, several stock cultures of the bacilli were placed in the refrigerator of a laboratory in London. The tubes were capped with rubber. As a consequence of the Great War, the cultures were not disturbed for almost six years and during most of this time the refrigerator was without ice. In the spring of 1920, the cultures were examined and it was found that one which had been grown on Dorset's egg medium was alive and, although feebly virulent, it incited sufficient lesions in a rabbit to preclude the possibility of the organism being of the human type.

Shope[8] reported on the survival of *Mycobact. tuberculosis* in suspensions in physiologic sodium chlorid solution which were kept in a refrigerator where the temperature was maintained about the freezing point. One strain of bovine origin and one of human origin were used. They had been grown on glycerin agar and were suspended in the proportion of 1 mg. of bacterial growth to 1 cc of 0.85 per cent sodium chlorid. Guinea pigs were used to determine viability. One animal received a dosage of 0.1 mg. and one received 1 mg. The suspensions were found to be

alive and virulent, even in the smaller dosage, after 310 days, 325 days, and 330 days. There was no discernible loss of virulence, although the maximal period of viability for these strains was not recorded.

Elimination of *Mycobact. tuberculosis* with the feces of tuberculous cattle has been considered a fruitful source of contagion ever since the work of Schroeder[9] proved that approximately 40 per cent of cows that reacted to tuberculin but which were, clinically, apparently healthy, ejected virulent bacilli with their excreta. Since the contamination of feed-lots, barns and pasture lands follows as a matter of course, the fate of the bacilli contained in these feces is of much significance in the pathogenesis of the disease. Briscoe,[10] in studying this problem, artificially infected cow manure with cultures of the bovine bacillus of tuberculosis and spread the material in layers 5 cm. thick. One part of these preparations was exposed to the direct sunlight and the other part was provided with shade. Viable and virulent bacilli were obtained from the material exposed to the direct sunlight for a period of approximately two months, and the virulence of the bacteria contained in the material that was protected from direct sunlight persisted for 73 days. Briscoe found also that the bacilli remained alive in running water for at least 441 days. The limit of the duration of viability under this condition was not determined.

Several significant and practical observations were made by Traum.[11] After a series of experiments to determine the resistance of the bacillus of tuberculosis of bovine origin to certain physical and chemical influences, Traum concluded:

> (1) The tubercle bacilli in feces and lung discharges can no longer produce tuberculosis in guinea pigs by inoculation when exposed in the dry season after three months, depending to a great extent upon how soon the vehicle in which the bacilli are found is freed from moisture, and (2) tubercle bacilli remain alive for at least six months in water. . . .

Williams and Hoy[12] studied the viability of the bovine form of *Mycobact. tuberculosis* on pasture land, in stored feces and in liquid manure. According to their report, the bacilli remained alive and virulent in feces exposed on pasture land in England for variable periods, depending on the time of the year.

The organisms retained their virulence for five months in winter, for four months in autumn, for two months in spring, and were nonvirulent after two months in summer. On pasture land infective feces that were protected from the direct sunlight contained virulent bacilli for four months in summer. Naturally infected feces of cattle stored in jars in a dark cool cellar remained virulent after twelve months, whereas artificially inoc-

ulated feces stored under the same conditions were virulent after 24 months. Artificially infected liquid manure, when stored underground in a 5-gallon jar, contained living and virulent bacilli for at least four months. Gradual diminution in the virulence of the organisms was noted.

That the bacillus of tuberculosis of bovine origin may retain its virulence for a considerable period when incorporated in dairy products is well illustrated in a study by Mohler, Washburn and Rogers.[13] Three samples of butter were utilized in the experiment. One sample of butter was made from milk to which the bovine bacilli were added before churning; another was made from the milk obtained from a cow affected with tuberculosis of the udder and which contained the organisms in large numbers, and the third was similar to the second except that the butter was unsalted. Salt in the proportion of 1 ounce to 1 pound of butter was added to the first two samples. The butter was placed in cold storage and portions were removed after different periods for the inoculation of guinea pigs. The results showed the bovine form of *Mycobact. tuberculosis* to be alive and virulent in all samples for at least 153 days. The maximal time was not determined.

The observations that have been made concerning the resistance of *Mycobact. tuberculosis* of bovine origin definitely distinguishes this bacterium as one of considerable tenacity. Low temperature, and a moist or wet environment, with protection from direct sunlight, are the factors of major importance in extending the longevity of this microörganism.

The results of the experiment forming the basis of this report are comparable with the previous data concerning the biology of the bovine form of the bacillus of tuberculosis. Since the material used consisted of 22 different strains of uncultured bacteria, the results should justify conclusions that might be untenable if only one or a few different strains had been used. The use of naturally infected material containing organisms that had been unaltered by previous residence on an artificial medium is a condition more closely approximating the normal than would be true had laboratory stock cultures been utilized.

Summary and Conclusions

The viability of different strains of *Mycobacterium tuberculosis* in naturally infected lesions of bovine tuberculosis is extremely variable. Twenty-two emulsions prepared by grinding lesions from as many different cattle in a solution of physiologic sodium chlorid and kept at a temperature of 5 to 10° C. were found in most instances to possess viable organisms that were

virulent for guinea pigs for approximately one year. After one year it was unusual for a strain to retain its viability, although a few strains were encountered that were capable of remaining virulent at ice-box temperatures for as long as 450 days. The limitation of the duration of virulence was not determined.

The structural integrity of the uncultured bacillus of tuberculosis of bovine origin is extremely stable under the conditions which obtained in this experiment, since there was no appreciable morphologic alteration detected in the acid-fast bacteria present in any of the emulsions. Although there is some evidence that certain strains of *Mycobact. tuberculosis* suffer progressive diminution of virulence after prolonged residence at ice-box temperatures, the exact status of this has not been determined.

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Trudeau Medal to Doctor Long

Dr. Esmond R. Long, of Chicago, received the Trudeau medal, yearly award for outstanding service to the tuberculosis movement, at the convention of the National Tuberculosis Association held at Colorado Springs during June. Dr. Long, at present a member of the faculty of the University of Chicago, is the discoverer of maioo, a new tuberculin for testing human beings and animals for tuberculosis. He is to resign his professorship at Chicago to become a director of the laboratory at the Henry Phipps Institute, Philadelphia, and associate professor of pathology at the University of Pennsylvania.

PRODUCTION OF AGGLUTININS FOR BRUCELLA ABORTUS IN CALVES, SWINE AND RABBITS BY SKIN AND MUCOUS MEMBRANE CONTACT*

By L. E. STARR, *Blacksburg, Virginia*
Department of Zoölogy and Animal Pathology
Virginia Agricultural Experiment Station

In testing sera, it has been found that certain animals, without previous history or symptoms suggestive of concurrent infection, at times show low-titre agglutination with *Brucella abortus*. This manifestation is frequently a temporary one, disappearing in a month or two. In some cases, it persists with slightly varying titre for several months.

Poppe[1] reports that agglutination tests made over a term of years on hundreds of animals, by numerous investigators, have established that infected animals generally show an agglutination titre of 1:100 and over and that it has been generally accepted that reactions of 1:100 are of positive value for Bang infection of cattle and 1:50 as of border-line value. Completely or partially positive reactions no higher than 1:25 are usually regarded as non-specific unless an increase in titre is shown on subsequent examination.

Successful infection of various species of animals by the organism, with subsequent agglutinin production, by subcutaneous, intravenous, intraperitoneal, urethral and oral routes, have been proven by numerous investigators. Schroeder and Cotton[2] report the successful infection of guinea pigs and cattle following application of the organism to the mucous membrane of the eye. Violle[3] infected guinea pigs with *Br. abortus* by rubbing a salve, containing the organism, into the epilated but unscarified skin. Hardy *et al.*[4] were able to infect a greater percentage of guinea pigs through the abraded skin, shaved skin and unaltered skin than by the mouth.

When infected animals are housed or penned with non-infected ones, there is abundant opportunity for contact with the specific microörganism in the soiled litter or for direct contact of animal to animal. The organism may gain entrance to the body through the gastro-intestinal tract with the feed, through the conjunctival sac from dust or fecal material, or directly through the skin.

In the light of experience with other diseases in man and animals, it is conceivable that such contacts are not necessarily followed by the development of frank clinical manifestations.

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Either because of the dosage-virulence factors on the part of the microörganism or the portal of entry factors on the part of the host, the organism may be gradually destroyed and the serum reaction the only evidence of the struggle that has taken place.

The object of this investigation is further to supplement studies that have already been made, by ascertaining the result of contacts such as conceivably might occur under actual conditions, with the production of agglutinins in rabbits, calves and swine.

Material and Methods

Recently isolated cultures of both bovine and porcine origin were secured from the B. A. I. Experiment Station, Bethesda, Md., the Kentucky Agricultural Experiment Station, and the Wisconsin Agricultural Experiment Station. In addition to these, a strain isolated from a human patient by Dr. Shaw, of Richmond Medical College, and a bovine strain isolated from the college herd, were used. The Richmond strain was typed by both Dr. Shaw and the author and found to be of the porcine variety.

Slants of beef-liver agar were sown with each of the different strains three days before the animals were to be treated. At the end of this time the slants were washed off with sterile normal saline solution. The four porcine cultures were mixed together and diluted to correspond with tube 5 of the McFarland nephelometer. The four bovine cultures were treated in the same manner. The experiment animals were treated immediately after preparation of the suspensions in the manner indicated in tables I, Ia, II, IIa, III, IIIa, IV and IVa.

For skin application, a spot on the neck just back of the ears was selected. This location was chosen because it was least accessible for the animals to wipe the organisms off and get them in the mouth. Except with those treated on the unaltered skin, an area three inches square was shaved. Half of those shaved were scarified with a scalpel until a slight hemorrhage resulted. Application of the organism was accomplished with a cotton swab that had been dipped in the suspension. Only enough of the suspension was used to wet the area prepared. Eye applications were made by brushing the moistened swab across the eyeball. Two pigs were fed two ounces each of the suspension of the organisms of bovine origin and two were fed two ounces of that of porcine origin. Two pigs, left untreated as controls, were kept in individual pens, with pigs on both sides that had been treated with bovine and porcine cultures respectively.

TABLE I—*Calves treated with strains of porcine origin.*

Ear Tag	Method of Treatment	Pre-Treatment Titres								Date of Treatment	Post-Treatment Titres											
		2–18–31				3–11–31					3–27–31				4–3–31				4–10–31			
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200
20951	Eye	0	0	0	0	0	0	0	0	3–11–31	0	0	0	0	0	0	0	0	1	0	0	0
20952	Unaltered skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0
20953	Shaved skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0
20954	Scarified skin	0	0	0	0	0	0	0	0		0	0	0	0	4	2	0	0	4	3	0	0

Ear Tag	Method of Treatment	Post-Treatment Titres																				
		4–17–31				4–24–31				5–5–31												
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200									
20951	Eye	1	0	0	0	2	0	0	0	0	0	0	0									
20952	Unaltered skin	0	0	0	0	0	0	0	0	0	0	0	0									
20953	Shaved skin	0	0	0	0	0	0	0	0	0	0	0	0									
20954	Scarified skin	4	0	0	0	0	0	0	0	0	0	0	0									

TABLE Ia—*Calves treated with strains of bovine origin.*

Ear Tag	Method of Treatment	Pre-Treatment Titres								Date of Treatment	Post-Treatment Titres											
		2–18–31				3–11–31					3–27–31				4–3–31				4–10–31			
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200
20955	Eye	0	0	0	0	0	0	0	0	3–11–31	0	0	0	0	0	0	0	0	0	0	0	0
20956	Unaltered skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0
20957	Shaved skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0
20958	Scarified skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0

Ear Tag	Method of Treatment	Post-Treatment Titres																				
		4–17–31				4–24–31				5–5–31												
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200									
20955	Eye	0	0	0	0	0	0	0	0	0	0	0	0									
20956	Unaltered skin	0	0	0	0	0	0	0	0	0	0	0	0									
20957	Shaved skin	0	0	0	0	0	0	0	0	0	0	0	0									
20958	Scarified skin	0	0	0	0	0	0	0	0	0	0	0	0									

TABLE II—*Calves retreated with strains of porcine origin.*

Ear Tag	Method of Treatment	Date of Treatment	Post-Treatment Titres																							
			6–10–31				6–18–31				6–27–31				7–9–31				7–17–31				7–24–31			
		5–5–31	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200
20951	Eye		0	0	0	0	1	0	0	0	1	0	0	0	2	1	0	0	0	0	0	0	0	0	0	0
20952	Unaltered skin		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20953	Shaved skin		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20954	Scarified skin		0	0	0	0	4	2	0	0	4	0	0	0	4	1	0	0	1	0	0	0	0	0	0	0

TABLE IIa—*Calves retreated with strains of bovine origin.*

Ear Tag	Method of Treatment	Date of Treatment	Post-Treatment Titres																							
			6–10–31				6–18–31				6–27–31				7–9–31				7–17–31				7–24–31			
		5–5–31	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200
20955	Eye		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20956	Unaltered skin		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20957	Shaved skin		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20958	Scarified skin		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

TABLE III—*Rabbits treated with strains of porcine origin.*

Ear Tag	Method of Treatment	Pre-Treatment Titres								Date of Treatment	Post-Treatment Titres																	
		2–25–31				3–18–31					4–18–31						4–29–31						5–20–31					
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200		1:25	1:50	1:100	1:200	1:400	1:800	1:25	1:50	1:100	1:200	1:400	1:800	1:25	1:50	1:100	1:200	1:400	1:800
15	Unaltered skin	0	0	0	0	0	0	0	0	3-18-31	4	4	0	0	0	0	3	0	0	0	0	0	0	0	0	0	0	0
16	Shaved skin	0	0	0	0	0	0	0	0		4	4	0	0	0	0	4	4	4	4	0	0	4	4	4	4	0	0
17	Scarified skin	0	0	0	0	0	0	0	0		4	4	0	0	0	0	4	4	4	4	4	4	4	4	4	4	4	4
18	Eye	0	0	0	0	0	0	0	0		4	0	0	0	0	0	4	4	3	0	0	0	4	4	4	4	4	0

TABLE IIIa—*Rabbits treated with strains of bovine origin.*

Ear Tag	Method of Treatment	Pre-Treatment Titres								Date of Treatment	Post-Treatment Titres																	
		2–25–31				3–18–31					4–18–31						4–29–31						5–20–31					
		1:25	1:50	1:100	1:200	1:25	1:50	1:100	1:200		1:25	1:50	1:100	1:200	1:400	1:800	1:25	1:50	1:100	1:200	1:400	1:800	1:25	1:50	1:100	1:200	1:400	1:800
19	Unaltered skin	0	0	0	0	0	0	0	0	3-18-31	0	0	0	0	0	0	0	0	0	0	0	0			*			
20	Shaved skin	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
21	Scarified skin	0	0	0	0	0	0	0	0		4	0	0	0	0	0	4	4	4	2	0	0	4	4	4	1	0	0
22	Eye	0	0	0	0	0	0	0	0		0	0	0	0	0	0	4	0	0	0	0	0	3	0	0	0	0	0

*Rabbit died.

TABLE IV—*Pigs treated with strains of porcine origin (May 5, 1931).**

| EAR TAG | METHOD OF TREATMENT | PRE-TREATMENT TITRES | | |

TABLE IVa—*Pigs treated with strains of bovine origin (May 5, 1931).*

Ear Tag	Method of Treatment	Pre-Treatment Titres		Post-Treatment Titres							
		4–3–31	5–4–31	5–15–31	5–23–31	5–30–31	6–10–31	6–17–31	6–24–31	7–1–31	7–10–31
		1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200	1:25 1:50 1:100 1:200
20925	Mouth	0 0 0 0	0 0 0 0	0 0 0 0	1 0 0 0	4 2 0 0	4 2 0 0	4 3 1 0	4 3 0 0	4 1 0 0	4 1 0 0
20913	Mouth	0 0 0 0	0 0 0 0	0 3 0 0	4 2 0 0	4 1 0 0	3 1 0 0	3 2 0 0	3 2 0 0	3 2 0 0	3 3 0 0
20926	Skin (sc)	0 0 0 0	0 0 0 0	3 0 0 0	3 2 0 0	4 4 3 2	4 4 1 0	4 3 1 0	4 1 0 0	2 0 0 0	2 0 0 0
20915	Skin (sc)	0 0 0 0	0 0 0 0	1 0 0 0	2 1 0 0	4 2 0 0	4 3 0 0	4 1 0 0	4 1 0 0	4 1 0 0	3 2 0 0
20927	Skin (sh)	0 0 0 0	0 0 0 0	2 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0
20917	Skin (sh)	0 0 0 0	0 0 0 0	0 0 0 0	1 0 0 0	2 2 0 0	3 2 0 0	3 2 0 0	0 0 0 0	0 0 0 0	0 0 0 0
20918	Eye	0 0 0 0	0 0 0 0	0 0 0 0	3 0 0 0	3 0 0 0	4 3 2 0	4 3 2 0	4 2 1 0	4 2 1 0	2 1 0 0
20919	Eye	0 0 0 0	0 0 0 0	0 0 0 0	4 4 2 0	4 3 1 0	4 0 0 0	2 1 0 0	2 0 0 0	0 0 0 0	0 0 0 0
20920	Skin (un)	0 0 0 0	0 0 0 0	0 0 0 0	0 0 0 0	1 0 0 0	2 0 0 0	1 0 0 0	1 0 0 0	1 0 0 0	1 0 0 0
20921	Skin (un)	0 0 0 0	0 0 0 0	1 0 0 0	0 0 0 0	2 0 0 0	2 2 0 0	3 1 0 0	3 1 0 0	2 0 0 0	2 1 0 0

Experiment Animals

Eight calves, four to five weeks old, were used. The status of the herds from which these calves were secured could not be learned. They were tested for abortion on arrival and gave negative reactions. Special wooden stanchions, spaced far enough apart so there could be no direct contact, were made for confinement of the calves. Three weeks following confinement in the stanchions, they were retested, found negative, and treated as shown in tables I, Ia, II and IIa.

Eight healthy negative rabbits were selected and placed in separate cages so that there was no contact between them. Following the second negative agglutination test, they were treated as shown in tables III and IIIa.

Twenty-two pigs, weighing about 75 pounds each, with unknown history, were used. They were tested for abortion on arrival. Three were positive in a 1:50 dilution and three partially. These six pigs were replaced by others that were negative. Individual pens 3′ x 7′ were constructed with boards closely fitted to a height of 12 inches from the floor with 4-inch spaces at intervals from that point to the top of the pens. The pens were provided with metal feed-troughs and were cleaned three or four times a week. Following the second negative abortion test, they were treated as shown in tables IV and IVa.

Summary

1. Application of live cultures of *Brucella abortus* (porcine) to the scarified skin of one calf and to the eye of another was followed by the appearance of low-titre agglutinins in the bloodstream. These persisted for a short time, disappeared and then reappeared after repetition of the same procedure. Two other calves to which the same cultures were applied, by way of the unaltered skin and shaved skin, respectively, reacted negatively.

2. The same experiment, repeated with bovine strains, resulted negatively in all four calves.

3. Application of suspension of live cultures of *Brucella abortus* (porcine) to the unaltered skin of one rabbit was followed by low-titre agglutinins; to the scarified skin, shaved skin and conjunctiva, it was followed by high-titre agglutinins.

4. Application of suspension of live cultures of *Brucella abortus* (bovine) to the scarified skin of a rabbit was followed by the appearance of high-titre agglutinins; to the eye, by low-titre agglutinins, and negative reactions to the unaltered and shaved skin.

5. A similar experiment with pigs, using suspensions of both

bovine and porcine origin, resulted in the appearance of agglutinins in low titre in all instances by all the methods of application. The agglutinins showed a tendency to decrease and disappear in about two months.

Conclusions

1. This study indicates that agglutinin production may result in calves, rabbits and swine following contact with *Brucella abortus* on the skin or conjunctival mucous membrane, abraded or unabraded.

2. Young calves are comparatively resistant to *Brucella abortus* infection.

3. Skin and mucous membrane contact may be responsible for many of the low-titre positive reactions found in cattle and swine.

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Broadcasting Program

Under the auspices of the Committee on Broadcasting of the New York State Veterinary Medical Society, the following talks were given during July, over Station WGY:

July 6—"An Abortion Control Plan," Dr. R. R. Birch, New York State Veterinary College, Ithaca.
July 13—"White Plague in the Poultry House," Dr. B. J. Cady, Albany.
July 20—"Destroying Lurking Enemies in the Milk Supply," Dr. J. F. Miller, State Department of Health, Albany.
July 27—"Heat Stroke," Dr. W. H. Kelly, Albany.

Weekly talks are scheduled to be given during August, as follows:

August 3—"Lumpy-jaw in Cattle," Dr. F. W. Andrews, Mount Kisco.
August 10—"The Importance of Parasites in Sheep-Raising," Dr. D. W. Baker, New York State Veterinary College, Ithaca.
August 17—"When Biddy Gets a Swelled Head," Dr. E. L. Brunett, New York State Veterinary College, Ithaca.
August 24—"Blackleg on the Farm," Dr. C. Linch, State Department of Agriculture and Markets, Albany.
August 31—"Foot Rot of Cattle," Dr. J. N. Frost, New York State Veterinary College, Ithaca.

The talks are given at 12:20 E. S. T., each Wednesday.

STUDIES IN CALFHOOD IMMUNIZATION AGAINST BANG'S DISEASE*

By A. L. DELEZ, *LaFayette, Indiana*
Department of Veterinary Science
Purdue University Agricultural Experiment Station

The possible value of calfhood vaccination as a means of developing immunity against Bang's disease was studied in a group of five heifer calves. They were injected intravenously with a strain of *Brucella abortus* which was pathogenic for guinea pigs. Each calf received the growth from two ordinary agar slants, incubated at 37° C. for 24 to 48 hours. The injections were administered by Dr. F. P. Mathews. The blood sera of this group were negative to the agglutination test previous to injection, when the calves were two to four weeks of age. A control heifer was allowed to run with the others until the fifth month of pregnancy. This calf was negative to the agglutination test, and was not injected at any time with *Br. abortus*.

Breeding took place when the heifers were between 12 and 15 months of age. Examinations were made on each animal to determine if pregnancy were established. Monthly agglutination tests of the blood sera were conducted throughout the greater part of the experiment. Serum dilutions of 1:20, 1:50, 1:100, 1:200, 1:500 and 1:1000 were used with an antigen of an approximate density of tube 3 of the McFarland nephelometer.

The dam of the bull used for breeding the heifers was a positive reactor to the agglutination test. At three months of age, the bull had a serum agglutination reaction of 1:500, when he was injected intravenously with the strain of *Br. abortus* used for the heifers. The agglutination reaction had dropped considerably at the third month following the injection and, when breeding services took place, it was approximately 1:50. He was slaughtered when 17 months of age. A bacteriological examination of the testicles, epididymides, and seminal vesicles did not reveal the presence of *Br. abortus* infection. This examination included guinea-pig inoculations. Neither gross nor microscopic lesions were found, but there was considerable evidence of spermatogenesis.

Heifers 930 and 947 were removed from the group and received intravenous injections of *Br. abortus* following the establishment of pregnancy. Heifer 930 received the growth from

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TABLE I—*Record of injections, serological data and breeding history of heifers on experiment.*

HEIFER	AGE AND DATE OF ORIGINAL INJECTION	AGGLUTINATION TITRE RECORD	AGE AND DATES WHEN BRED	DATE AND METHOD OF EXPOSURE	PARTURITION RECORD
927	4 weeks 5–28–28	Before injection: −20. *1929:* June, 1000; July, 500; Aug., 100; Oct., 25; Dec., 25. *1930:* Feb., 25; June, 100; July, 50; Sept., 1000; Oct., 1000; Nov., 500; Dec., 100. *1931:* Jan., 200; March, 1000.	15 mos. 8–7–30	None	1–16–31, aborted fetus 17 in. long. Retained placenta.
928	2 weeks 5–28–29	Before injection: −20. *1929:* June, 200; Aug.-Dec., 500. *1930:* Feb.-June, 100; July, 500; Sept., 1000; Oct., 500; Nov., 200; Dec., 100. *1931:* Jan., 100; March, 200; May-June, 200; Sept., 50.	14–15 mos. (1) 7–29–30 (2) 8–20–30	None	6–5–31, a full-term fetus removed manually. Fetus dead. Extensive necrosis of cotyledons.

930	4 weeks 7–31 29	Before injection: −20. *1929:* Aug., 100; Oct.-Dec., 50. *1930:* Feb.-Sept., 100; Oct.-Dec., 20. *1931:* Jan., 500-1000; Mar., 1000.	11 mos. (1) 5–11–30 (did not conceive) (2) 8– –30	12–12–30, growth of 2 agar slants intravenously.	2–1–31, aborted fetus 17 in. long. Placenta expelled. Necrotic cotyledons.
933	3-4 weeks 7–31–29	Before injection: −20. *1929:* Aug., 200; Oct. '29-July '30, 100; Sept.-Nov., 200; Dec., 50. *1931;* Jan.-May, 20.	13 mos. 8– –30	None	4–29–31, gave birth to live calf, somewhat small. Retained placenta.
947	2 weeks 7–31–29	Before injection: −20. *1929:* Aug.,-20; Oct., 100; Dec., 25. *1930:* Feb.-June, 25; July-Nov.,-20; Dec., 100-500. *1931:* Jan., 1000.	13 mos. 7–16–30	12–8–30, growth of 1 agar slant intravenously.	1–1–31, aborted fetus 17 in. long. Retained placenta.
936	Control (not injected)	Aug. '29-June '31, −20.	14 mos. 8–27–30	None	5–30–31, normal parturition. No lesions.

two agar slants, when pregnant about four months, and heifer 947 received the growth from one agar slant one month later in pregnancy. No exposures were given to the other heifers during gestation. After heifer 936 (control) calved normally, she was allowed to associate with two infected heifers in a pasture-lot, in which one of the heifers aborted.

Following abortion or calving, bacteriological examinations, including guinea-pig inoculations, were made of the placentæ and colostrums, when available, and fetal tissues. At autopsy, uterine and udder tissues were obtained for pathological and bacteriological examinations. The tissues were fixed in formol and stained by the hematoxylin-eosin method.

Heifers 930 and 947 failed to show much antibody formation following the calfhood injections and their titres remained low until they were exposed during pregnancy. The first month subsequent to injection, heifer 927 had a high agglutination reaction. It soon dropped, however, until the sixteenth month of age, when it arose to a high level again. The titre fell to 1:100 at the nineteenth month, followed by a slight rise at the twentieth month, when abortion occurred, after which it quickly regained its former level. Heifer 928 showed a rise of agglutination titre between the third and fifth months, and heifer 933 at the seventh month of age. Except for a high titre in heifer 928 at the sixteenth month, there was a low reaction in both cases throughout the experiment. Control heifer 936 remained negative to the agglutination test and developed a low titre only after she was kept in the pasture-lot with the infected heifers.

Abortions occurred in heifers 930 and 947 following the intravenous exposures. Both animals aborted at the same period of pregnancy. As noted above, heifer 947 received a much smaller amount of infection than heifer 930. Heifer 947 aborted 24 days after the exposure and heifer 930 carried her fetus 51 days following the intravenous injection. Uterine infection was demonstrated in each case and the colostrum of heifer 947 was positive.

Heifer 927 aborted at the 162nd day of gestation. No artificial exposure was given. *Br. abortus* was isolated from the fetus and colostrum. Due to the putrefactive changes in the placenta following retention, no bacteriological examination was made of this material.

Heifers 928 and 933 carried their fetuses to a full term. The fetus of heifer 928 was dead when delivered on the 289th day of gestation. The fetal membranes of both heifers showed extensive necrosis of the cotyledons. *Br. abortus* infection was not demonstrated in either case.

Control heifer 936 calved normally on the 276th day of gestation. No infection was revealed at the time of parturition.

The injected heifers were killed at periods of 19 to 96 days following abortion or calving, as indicated in table II. Involution of the uterus was complete or nearly complete in all the heifers at autopsy. *Br. abortus* was not found in the uterus of any of the heifers. However, it was isolated from the udders of the three heifers that aborted (927, 933 and 947). Control heifer 936 was killed 103 days following parturition and approximately 50 days subsequent to her exposure to the aborting heifer in the pasture-lot. *Br. abortus* infection was found only in a guinea-pig inoculation of the supramammary lymph-glands.

There were no microscopic alterations found in the uteri of four of the injected heifers nor in control heifer 936. The uterus in heifer 947 was not obtained for study. A diffuse interstitial mastitis was revealed in the udders of the heifers from which *Br. abortus* was isolated (927, 933 and 947). This was indicated by a proliferation and cellular infiltration of the interstitial tissue.

Discussion

It was anticipated that a rather severe exposure would be necessary to induce abortion in heifers 930 and 947, since their blood sera gave only slight agglutination reactions to the calfhood injection. Both animals, however, failed to resist intravenous exposure. Buck,[1] in a calfhood vaccination experiment, found that one of five heifers, receiving a vaccine prepared from strains of *Br. abortus* that had been under artificial cultivation longer than strains in other vaccines used in the experiment, failed to withstand oral exposure.

The abortion in heifer 927 may indicate the infection resulted from the calfhood vaccination, or the lack of immunity to an exposure that would be expected to occur under natural conditions. Buck[1] states further that one of three heifers, which were given a vaccine considered the most virulent of the three types he used, apparently developed udder infection.

Although heifers 928 and 933 carried their calves to a full term, the fetal membranes in both cases showed lesions similar to those of *Br. abortus* infection. While the organism was not isolated from either of these heifers, they would necessarily be considered dangerous cases clinically.

Heifer 936 (control) apparently contracted infection only after her exposure following parturition.

The lesions found in the udders from which *Br. abortus* was isolated were similar to those described by Hallman, Sholl, and Delez,[2] Hallman and Delez,[3] and Sholl and Torrey.[4] Lesions of

TABLE II—*Bacteriological data of heifers on experiment.*

HEIFER	EXPOSED	ABORTED	BRUCELLA ABORTUS ISOLATED			AUTOPSIED (DAYS AFTER ABORTION OR CALVING)	BRUCELLA ABORTUS ISOLATED	
			COLOSTRUM	FETUS	PLACENTA		UTERUS	UDDER
927	No	Yes	+	+	0	4–14–31 (88)	−	+
928	No	No	−	−	−	9–9–31 (96)	−	−
930	Yes	Yes	0	+	+	4–14–31 (72)	−	+
933	No	No	−	−*	0	5–18–31 (19)	−	−
947	Yes	Yes	+	+	+	2–5–31 (35)	0	+
936	No (Control)	No	−	0	0	9–10–31 (103)	−	+†

0 = no examination.
*Examination of tissues of calf 6 days of age.
†Isolated from supramammary lymph-glands following exposure subsequent to normal parturition.

this type involving all quarters of the udder would in all probability seriously interfere with the functional activity of the organ.

SUMMARY

The results obtained in bred heifers which had received intravenous injections of *Brucella abortus* during the first month of age are as follows:

Two heifers, which gave only slight response to the calfhood injection, as indicated by the blood agglutination titres, aborted when given an intravenous exposure of a virulent strain of *Br. abortus*. The infection was revealed in each case at the time of abortion and also in the udders at autopsy.

One heifer, which received no further exposure following the calfhood injection, aborted. Uterine and udder infection with *Br. abortus* was demonstrated.

Two heifers, which also received only the calfhood injections, carried their calves to a full term. At parturition there were gross lesions suggestive of *Br. abortus* infection in both cases. Bacteriological examination failed to show the presence of the organism in either case.

An uninjected control heifer was allowed to associate with the injected animals, and calved normally. Later she was found susceptible.

A diffuse interstitial mastitis was observed in the heifers from which *Br. abortus* was isolated.

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Tuberculosis Eradication Survey

A booklet entitled "Ridding the Nation of Tuberculosis in Live Stock," by H. R. Smith, Live Stock Commissioner of the National Live Stock Exchange, Chicago, Ill., outlines the progress made to date, and contains charts showing the status of various parts of the United States as regards eradication.

Eastern Iowa Clinic Postponed

The Executive Council of the Eastern Iowa Veterinary Association has postponed the date of the clinic, originally scheduled for August 18, until September 6, 1932. The clinic will be held at the sale barn of Dr. F. M. Wilson. at Mechanicsville, Iowa.

CATTLE AS A POSSIBLE SOURCE OF INFECTION FROM BRUCELLA ABORTUS VAR. SUIS*

By AMANDA HELEN HARMS, *Lexington, Ky.*
Kentucky Agricultural Experiment Station

In the year 1925, an aborted fetus from a heifer that had been shipped from Tennessee to Kentucky was brought to this laboratory. The owner of this heifer had had no abortions on his farm and thought the abortion was due to accident. The internal organs of the fetus were cultured and *Brucella abortus* C_1 was isolated from the umbilical vein, from the lung, and from the stomach contents of the fetus. This organism grew much like the bovine strains of abortus,[1] in that ten successive subcultures were required before it adapted itself to aerobic growth, in contrast to *Brucella abortus* var. *suis* which, in our experience, grows aerobically in first cultures when isolated from aborting sows.[2] In appearance it resembled *Br. abortus* var. *suis*. When grown on agar, colonies of porcine strains are more opaque and granular than bovine strains, which are clear, moist and more raised. Some of the bovine strains have a few granules. When colonies of porcine strains and strain C_1 were imbedded in agar, concentric rings, sometimes two, sometimes three and sometimes four, were observed and areas became darker toward the centers of the colonies. Most of the imbedded colonies of bovine strains used for comparison were clear. One or two concentric rings were noted in a few of the colonies, but they were all lighter in color than the porcine strains.

An abundance of hydrogen sulfid gas was produced by a porcine strain (M) and strain C_1, as shown by the blackening of lead acetate agar. A bovine strain (C) also produced hydrogen sulfid but not so abundantly as the other strains. Both urea and asparagin were decomposed, with the production of ammonia. The reaction in asparagin medium, however, was slight with all three strains. Ammonia was produced in nitrate peptone broth with the three strains. There was a slight reduction of nitrate to nitrite with the bovine strain C, but the porcine strain M and the strain C_1 showed no reduction.

Several crystals, as described by Huddleson and Winter,[3] were formed in agar with the bovine strain, none with strain C_1, and only one with the porcine strain.

*The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director. Received for publication, February 8, 1932.

When compared by using gentian violet[4] (table I), and by using Huddleson's dye plates[5] (table II), strain C_1 was shown to be of the porcine species.

TABLE I—*Growth behavior toward gentian violet.*

STRAIN	1:50,000	1:100,000	1:250,000	CONTROL
M (porcine)	0	0	3	4
C (bovine)	1	3	4	4
C_1	0	0	3	4

0 = no growth.
1 = very scanty growth.
2 = scanty growth.
3 = moderate growth.
4 = abundant growth.

TABLE II—*Growth on Huddleson's dye plates.*

STRAIN	METHYL VIOLET 1:100,000	BASIC FUCHSIN 1:50,000	THIONIN 1:50,000	CONTROL NO DYE
M	1	0	4	4
C	4	4	0	4
C_1	1	0	4	4

The agglutinin-absorption test was employed as a means of serological classification. In this test strains C_1, C (bovine) and M (porcine) were employed. All these strains were isolated by this laboratory: C_1 from an aborting cow, C from an aborting cow and M from an aborting sow. Rabbits were injected with strain C for bovine serum C and with strain M for porcine serum M. Agglutination reactions with antigen C_1 before and after absorption tests, as shown in table III, showed that it contained agglutinins specific for the porcine strains of *Br. abortus*. Table III shows that the type of strain C_1 was identical with that of the porcine strain M.

At the Connecticut (Storrs) Station,[6] in three instances, strains of undoubted bovine origin have given the characteristics of *Br. abortus* var. *suis* or *Br. melitensis*. At the Illinois Station,[7] experimental infection of a heifer by intravenous injection with porcine strains was followed by abortion.

In our routine testing of bovine samples of blood for Bang's disease, a few cows that were known to have aborted failed to respond to the agglutination test when bovine antigens were used, but gave positive reactions when tested with an antigen

made from C_1. Possibly these cows were infected with the porcine variety of *Br. abortus*. As an example, the serum from a cow that aborted recently and responded negatively to the test was sent to this laboratory with a request that it be tested with several antigens. Twenty-one bovine antigens, six melitensis antigens, an antigen made from a human strain, an antigen made from porcine strain M and an antigen made from C_1 were used. Results were negative with all but the two last-named antigens. The serum was positive in a 1:50 dilution with C_1, and in a 1:200 dilution, with 50 per cent in a 1:500 dilution with the porcine strain M. These tests indicate that cattle sometimes may become naturally infected with porcine strain of *Br. abortus*.

TABLE III—*Agglutinin absorptions with strain* C_1.

SERUM	CONDITION OF SERUM	ANTIGEN	DILUTION				
			1:100	1:200	1:400	1:1000	1:2000
Bovine C	Not absorbed	C	4	4	4	4	4
	Absorbed by C	C	0	0	0	0	0
		C_1	0	0	0	0	0
	Absorbed by M	M	0	0	0	0	0
		C_1	0	0	0	0	0
	Absorbed by C_1	C	4	4	4	4	4
		M	0	0	0	0	0
		C_1	0	0	0	0	0
Porcine M	Not absorbed	M	4	4	4	4	4
	Absorbed by C	C	0	0	0	0	0
		C_1	4	4	4	4	4
	Absorbed by M	M	0	0	0	0	0
		C_1	0	0	0	0	0
	Absorbed by C_1	C	0	0	0	0	0
		M	0	0	0	0	0
		C_1	0	0	0	0	0

4 = complete sedimentation.
2 = 50 per cent sedimentation.
0 = no sedimentation.

SUMMARY

An organism isolated from the aborted fetus of a heifer was identical with bovine strains in that it required several successive cultures before it adapted itself to aerobic growth. In all other tests it was identical with *Brucella abortus* var. *suis*.

ACKNOWLEDGMENT

The writer is grateful to Dr. Philip R. Edwards, of the Department of Animal Pathology, for valuable advice and aid in making the agglutinin-absorption tests.

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ON TO ATLANTA

This monument in downtown Atlanta was erected in honor of Henry Grady, eminent southern author and editor, who was equally loved by North and South.

FATAL RESULTS IN GUINEA PIGS FOLLOWING INTRA-ABDOMINAL INJECTION OF FRESH COW SERUM*

By RONALD GWATKIN, *Toronto, Canada*
Department of Veterinary Science, Ontario Research Foundation

In preliminary experiments with the sera of two cows that were being immunized against *Brucella abortus*, it was observed that the fresh serum in doses larger than 1 cc proved fatal for guinea pigs by the intra-abdominal route.

Symptoms appeared in from 1½ to 2 minutes after an intra-abdominal injection of 5 cc. Violent jerking movements of the head and body persisted as a rule for from 4 to 5 minutes. The animals then became quiet and lost the use of the hind quarters in about 20 minutes, lying quietly until they died one to two hours after injection. The mucous membranes of the head became very pale. In white guinea pigs this was plainly seen in the eyes. When smaller doses or larger guinea pigs were employed, death was delayed for several hours. Those animals injected with 1 cc showed nervous symptoms but recovered. Subcutaneous injection of serum gave rise to slight or no nervous manifestations, but there was a marked local reaction followed by sloughing of the skin. In some earlier experiments,[1] fresh cow serum was used without fatal results. Symptoms may have appeared but, as no trouble was anticipated, the animals were not kept under observation following the injection of serum, and the following morning all appeared healthy.

Postmortem examination showed a severe congestion of the abdominal organs and peritoneum. The small intestines contained a large amount of laked blood and there were multiple hemorrhages in the liver and spleen. The injected serum was stained with hemoglobin. Such blood as remained in the circulation appeared to clot normally and the serum was slightly or not at all stained with hemoglobin.

Fresh cow serum was added to 1 cc of a suspension of washed guinea-pig cells in 0.85 per cent NaCl solution in quantities of 0.01, 0.1 and 1.0 cc, and placed in a water-bath at 37° C. The largest dose produced hemolysis of the red cells in one minute, 0.1 cc in ten minutes, while 0.01 did not produce hemolysis. With some samples this tube was completely hemolysed after standing at room temperature overnight, while the controls were unchanged. Serum heated at 56° C. for 30 minutes did not produce

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hemolysis and was harmless when injected. Samples of serum, which had caused death in 2-cc doses when fresh, produced only slight nervous symptoms when a dose of 5 cc was injected nine days after collection and were not hemolytic *in vitro*.

Fifty-three samples of cow serum were examined when 48 hours old by the addition of 0.5 cc of serum to 1 cc of a 1 per cent suspension of guinea-pig cells. All produced hemolysis. Six samples required from 5 to 10 minutes, the remainder produced complete laking in 3 minutes. Another group of 86 cow sera were similarly examined. Four samples required from 4 to 14 minutes to produce complete hemolysis. The remainder went within 3 minutes. Two samples that produced hemolysis in 1½ minutes were combined and also two that required 10 and 14 minutes. A guinea pig was injected with 5 cc of each mixture. The animal that received the strongly lytic serum was severely affected and died in 75 minutes. In the guinea pig that received the less hemolytic serum, symptoms were much less severe and the animal made a rapid recovery. Another group of 40 samples contained three which did not produce complete hemolysis after incubation for forty minutes.

Fresh cow serum hemolysed human, rabbit and sheep cells, but was somewhat slower, requiring from 4 to 5 minutes. No laking occurred in the 0.01-cc tube overnight. Heated cow serum was not reactivated by the addition of fresh guinea-pig serum. Guinea-pig serum produced laking of cow cells in the 0.1-cc tube but not in the 0.01-cc tube. Horse serum lysed guinea-pig cells when 0.5 cc was added to 1 cc of suspension but not in a higher dilution.

In summarizing the above experiments it is evident that fresh cow serum cannot be injected into guinea pigs without danger. If the serum is heated at 56° C. for 30 minutes or kept until the hemolytic activity is lost, which occurred in nine days in these experiments, it is innocuous for these animals.

ACKNOWLEDGMENT

I am indebted to Drs. D. T. Fraser and N. E. McKinnon, of the School of Hygiene, and to Drs. H. B. Speakman and S. Hadwen for their interest and assistance.

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The Anderson Veterinary Clinic has been incorporated at Anderson, Ind., with a capital stock of 1,000 shares of no par value. The incorporators are Dr. J. H. Blake, Dr. R. E. Speck and Robert Blake.

TWO CASES OF INFESTATION OF THE DOG WITH DIOCTOPHYME RENALE*

By CARL OLSON, *Rochester, Minnesota*
Division of Experimental Surgery and Pathology
The Mayo Foundation

Dioctophyme renale, commonly known as the giant kidney worm, is the largest of the true nematodes. It is reported to occur in the marten, dog, fox, wolf, coyote, horse, ox, and rarely in man. Its occurrence in these animals is uncommon, with the possible exception of the mink. The adult parasite usually is in

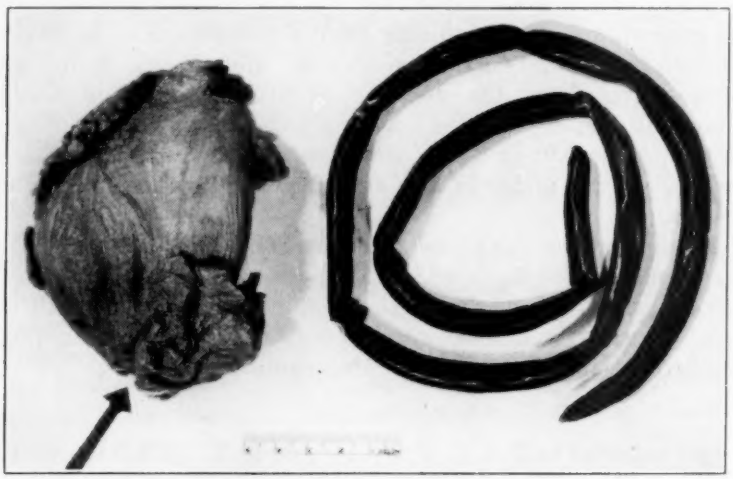

FIG. 1. Urinary bladder (case 1) and *Dioctophyme renale;* female. The arrow points to the perforation at the apex.

the kidney, bladder or peritoneal cavity. The life cycle is unknown, although certain fish have been suspected as the intermediate host. Feeding the fertilized ova to the dog has given negative results.[1] I shall report here two cases of *Dioctophyme*

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renale in the dog. One of these cases is of particular interest since the dog was infested with three of the parasites, and manifested little if any clinical evidence of the infestation.

Case 1: The dog was a male, adult, mongrel bull terrier. The

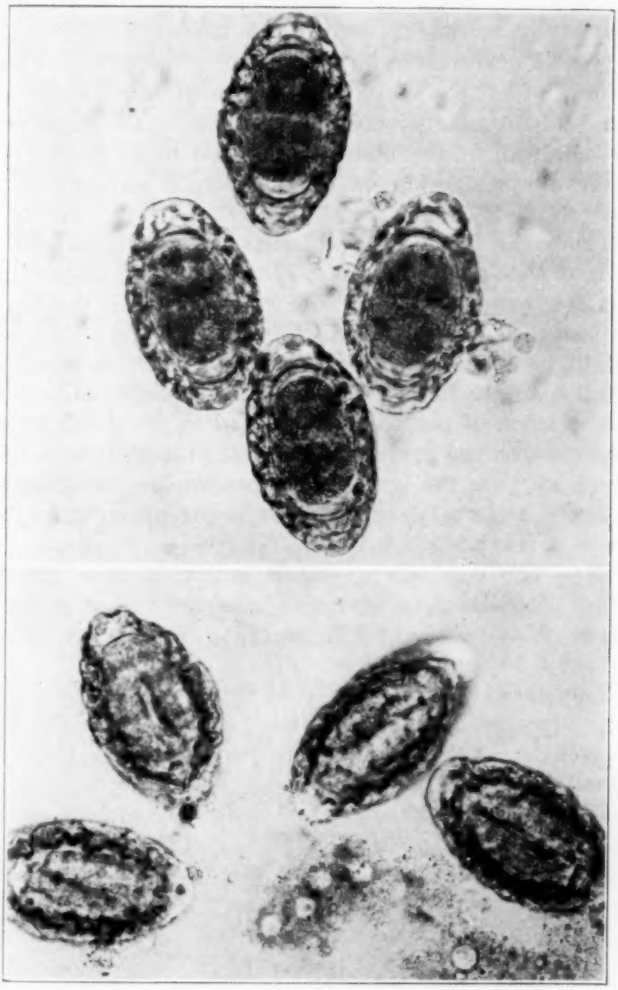

FIG. 2 (above). Ova of *Dioctophyme renale* (case 2) in the two-cell stage as found in urine (x 400).

FIG. 3 (below). Embryonated ova (case 2) 68 days after they had been obtained from the urine (x 400).

definite origin and the history of this animal were not available; however, it was ascertained that the dog was raised in the vicinity of Rochester, Minnesota. It was secured in December, 1930, and first used experimentally in February, 1931, when it under-

went a series of experiments on wound healing. During this time there were no indications of infestation with this parasite.

July 14, 1931, the dog was found dead in his kennel. At necropsy a large round worm was found lying free in the peritoneal cavity. A large quantity of sanguineous fluid, estimated at about 4 liters, was present. At the apex of the urinary bladder was a perforation about 75 mm in diameter. There was pyelitis of the left kidney. The mucosa of the bladder was excoriated, and the bladder contained a few small urinary calculi. Grossly, the wall of the bladder appeared to be thick; histologically, there was erosion of the epithelium, congestion of the blood-vessels, and hemorrhages into the tissue spaces. The parasite found in the peritoneal cavity was identified as a female specimen of *Dioctophyme renale*, 77 cm long and 15 mm in diameter. A few eggs of the parasite were found in the fluid from the peritoneal cavity (fig. 1.),

It might be assumed that the parasite had inhabited the urinary bladder of the host, without causing recognizable symptoms until perforation of the bladder resulted in the death of the animal. As regards the pyelitis of the left kidney, it is a matter of conjecture whether the larval parasite invaded the urinary tract at this point and then passed through the ureter into the bladder where it reached maturity.

Case 2: The dog was a female, adult, brindle bull terrier. The owner stated that the dog was born and raised in the region of Milaca, Minnesota, and brought to Rochester, Minnesota, when about a year old.

The animal was assigned for experimental surgery, and laparotomy was performed in October, 1931. When the abdomen was opened the mesentery was found to be inflamed and covered with small reddish stippling.

A male parasite, *Dioctophyme renale*, 32 cm long and 5 mm in diameter, was found in the region of the liver and lesser omentum (fig. 2). A mass of what appeared to be connective tissue was attached loosely to the mesentery near the pancreas. A cover-slip preparation of a portion of this mass revealed the presence of ova of *Dioctophyme renale*. Histologic section disclosed organized granulomatous tissue surrounding the eggs. Further examination of the abdomen revealed that the right kidney was very much enlarged, and the renal pelvis filled with some solid material. The presence or absence of a female parasite in the peritoneal cavity could not be definitely ascertained; the results of search were negative. The abdominal wound was closed after removal of the male parasite and a portion of the mass of eggs. After the dog recovered from the operation, the

urine was examined for the presence of eggs. Ova of *Dioctophyme renale* were found in great numbers. These ova appeared to be in the two-cell stage. Specimens of this material have been observed at various times over a period of several months, with the result that embryonation of the eggs has been observed, thus indicating that both a female and a male parasite are present in the right kidney of this dog (figs. 3 and 4).

The dog has been held for further study. Thus far, slight hematuria at irregular intervals, together with the constant presence of eggs in the urine, have been the only evidence of infestation. The general condition of the animal remains good. On palpation of the abdomen pain is not evidenced, nor can the enlargement of the right kidney be detected. The latter may be due principally to the obesity of the animal.

REFERENCE

[1]Lukasiak, Jacob: Anatomische und Entwicklungsgeschichtliche Untersuchungen an Dioctophyme renale (Goeze 1782). (Eustrongylus gigas Rud.) Arch. d. Biol. d. Soc. des Sc. et des Lettres de Varsovie, iii (1929), pp. 1-100.

HYGROMA OF THE KNEE

By HARRY H. ROSS, *Brandon, Man.*

Subject: Shorthorn cow, Roan Lady 11th 210449, bred by George Rankin and Sons, the oldest continuous breeders of Shorthorn cattle in the Dominion of Canada.

Diagnosis: Hygroma of the knee.

Operation: On September 21, 1931, an operation was performed to relieve the condition, as the animal was considered

FIG. 1. Shorthorn cow before and after operation for hygroma of the knee.

valuable enough to warrant surgical intervention. The entire hygroma, together with its indurated wall, was completely excised. Considerable care was necessary to avoid injuring the synovial sheaths of the extensor tendons. The operation was performed under anesthesia, and was completed by firmly suturing the lips of the wound, and applying an antiseptic surgical dressing. The patient was prevented from lying down until the wound had firmly united.

The hygroma as removed weighed 18 pounds 1 ounce. The cornification of the skin and the calcification and ossification of the walls was very extensive, as the result of the chronic condition of the process. The value of the cow alone warranted the operation. Mr. George Rankin, son of Mr. John R. Rankin and junior student at the Ontario Veterinary College, assisted me with the operation.

A DISEASE AFFECTING THE LEGS OF CATTLE*

By L. P. DOYLE, *La Fayette, Ind.*
Purdue University Agricultural Experiment Station
Department of Veterinary Science

During the late summer and fall of 1931, a disease was observed to be affecting particularly the front legs of cattle in Indiana. The majority of the cases occurred in the southern portion of the State. Some instances, however, were reported as far north as Miami County. There was said to have been as many as 500 cases in one of the southern counties. The opinion was quite general, among farmers, at least, that this was a new disease. No doubt the affection has rarely, if ever, occurred as extensively as it did last fall, but sporadic cases have very likely been occurring for a number of years.

Some of the affected animals were "family cows," on small farms where only one cow was kept; others were in herds comprising a dozen or more cattle. In the latter herds, the number of animals affected varied from one to many.

Cause: The precise cause of the disease was not definitely determined. The opinion was quite general among farmers and veterinarians that flies played an important part in producing the disease. The term "fly poisoning" was frequently used by farmers in referring to the condition. The high incidence of the disease during the season when biting flies were most numerous, and the preponderance of lesions in the front legs

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would, of course, give support to the opinion that flies played an important part in causing the disease.

Symptoms: Usually the first symptom noted by the owners of the affected cattle was swelling in the coronary region or pastern. Later the swelling extended higher up the limb, usually finally reaching the carpal region or a variable distance above. In some instances the swelling extended up into the brisket and shoulder. The skin of the volar surface of the affected limb cracked horizontally and exuded a serous fluid, which occasionally became purulent (fig. 1). In some cases rather large plaques of the skin become loosened and could be pulled off easily. Well-marked swelling of the prescapular lymph-gland

FIG. 1 (left). Case showing fissures in the skin of the front legs
FIG. 2 (right). Case showing marked swelling of the right prescapular lymph-gland and the surrounding tissue.

on the affected side was commonly found (fig. 2). The precrural lymph-gland also was sometimes enlarged, even though there was no noticeable affection of the corresponding hind limb. Some veterinarians reported finding vesicles and nodules in the buccal mucous membrane of animals which showed leg symptoms. The mortality rate was usually low. The fatal cases usually occurred in animals which developed extensive swelling of the brisket and shoulder. Some veterinarians believed that the evidence of lymphadenitis persisted for some time after the other symptoms had subsided. The length of the troublesome

period of the disease in individual animals was, as a rule, not longer than two or three weeks.

Inoculation experiments: Some of the sloughed skin, as well as exudate and scrapings from recent lesions, were triturated in physiological salt solution and then rubbed on shaved and scarified areas of the skin of guinea pigs and two cows. No significant results were obtained. A guinea pig, which was injected subcutaneously with some of the triturated material, became moribund within three days and was killed. Autopsy showed some subcutaneous edema at the point of inoculation and numerous yellowish-gray foci in the liver. An apparently pure culture of *B. pyocyaneus* was obtained from the liver. This culture proved to be highly pathogenic for guinea pigs when injected either subcutaneously or intraperitoneally. The growth (24 hours) from one agar slant was injected under the skin of a mature cow. A large painful swelling developed within three days, lasted for about six days and then subsided without the occurrence of any other noticeable symptoms.

The etiological significance of *B. pyocyaneus* in this disease of cattle is problematic. It is a well established fact, of course, that *B. pyocyaneus* is definitely pathogenic for certain laboratory animals. Moreover, there is no reason to question its etiologic rôle in some disease processes in the human. It is interesting to note that Ehlers[1] reported two human cases in which there was vesicle formation and from which *B. pyocyaneus* was isolated.

ACKNOWLEDGMENT

The writer is indebted to Dr. F. H. Brown, State Veterinarian, and members of his staff for considerable information regarding the symptoms and distribution of this disease.

REFERENCE

[1]Ehlers: Cited by Park and Williams: Pathogenic Microörganisms.

AN INTERESTING CASE OF NYMPHOMANIA IN A VIRGIN HEIFER*

By C. F. CLARK, *East Lansing, Mich.*
Michigan Agricultural Experiment Station

While cases of cystic ovary and nymphomania occasionally occur in sterile dairy cows, the writer believes that typical cases of nymphomania in virgin heifers are relatively infrequent.

Subject: A registered Ayrshire heifer, born November 22, 1929.

History: Normal growth and development had proceeded up

*Received for publication, July 14, 1932.

to March, 1931. The heifer had had a few estrual periods at normal intervals, but had not been bred. About the last of February, 1931, it was noticed that the mammae were undergoing considerable enlargement and development. It was suspected that some of her mates might be stimulating such development by sucking, but observation did not confirm this. It was then suspected that the heifer had been bred accidentally, as several months before, a young bull had broken out of his pen and been loose in the barn during the night.

Case Report

March 10, 1931: Examined and found not pregnant; no recognizable abnormalities of genitalia, other than marked udder development.

April 14, 1931: Heifer has begun to bellow continuously while in exercise-yard; paws the ground frequently, mounts her fellows and simulates coitus; sacrosciatic ligaments appear to be relaxing. Does not evidence estrum itself. Examination revealed a corpus luteum in the left ovary. The right ovary contained what appeared to be a small cyst, which was crushed.

May 7, 1931: The same chain of symptoms above noted has persisted. The tail-head is decidedly elevated and tilts forward. Examination reveals the left ovary of normal size, although harder than normal. The right ovary contained a cyst, ¾ to 1 inch in diameter. This cyst was ruptured and the ovary massaged thoroughly.

May 18, 1931: Same symptoms as previously noted. Left ovary normal in size, although harder than normal. Right ovary contained two cysts, ¾ inch in diameter. These cysts were ruptured and the ovary massaged thoroughly.

May 24, 1931: Estrum evident. Symptoms of nymphomania have abated. Was not bred.

June 12, 1931: Estrum. Bred.

July 7, 1931: Estrum. Bred. Tail-head nearly normal in appearance.

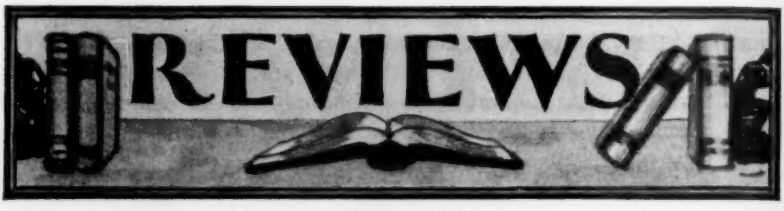
July 26, 1931: Estrum. Bred. Tail-head appears normal.

August 14, 1931: Estrum. Bred.

September 18, 1931: Left uterine horn slightly enlarged. Left ovary contains corpus lutem. Right ovary negative. Early pregnancy?

October 13, 1931: Pregnant. Left uterine horn enlarged and fluctuating, left ovary contains corpus luteum.

May 24, 1932: Delivered a normal male calf, weight 76 pounds; gestation, 283 days.



KLAUENKRANKHEITEN. (Diseases of the Claws.) E. Hess. 3rd edition, completely revised by Dr. Ernst Wyssmann. 255 pages. Illustrated. Urban & Schwarzenberg, Berlin and Vienna, 1931.

This is the third part of Volume IV of the Handbook of Veterinary Surgery and Obstetrics, which is edited by Drs. Eugen Fröhner and Theodor Schmidt. The previous edition of this standard work by the late Dr. E. Hess was published eighteen years ago. Dr. Hess has an excellent reputation, not only abroad but also in this country. He was one of the foremost veterinary practitioners, and his death in 1920 was most untimely.

The book is divided into eight parts. Part one is by Dr. T. O. Rubeli, and is a discussion of the anatomy of the feet of cattle, with special reference to the claws. Part two, also by Dr. Rubeli, is on the general form of the claws, and discusses abnormalities. The third part is on the care of the feet. The fourth is on means of restraint. The fifth, sixth and seventh parts are devoted to short discussions concerning the general structure of the front and hind claws and operations on the claws. The eighth part, which makes up the greater portion of the book, is devoted largely to a discussion of the diseases affecting the feet of cattle.

The text is illustrated with excellent drawings and photographs. The discussion of the diseases is admirably lucid, and the descriptions of methods are readily understandable. It is surprising that the method of stretching the animal between two posts is not illustrated, as this method is not uncommonly employed in this country for operations upon the claws.

Unfortunately, up to this time, this book is available only in the original German text. There is very little literature on diseases of the claws from American authors. This is a relatively important branch of veterinary practice and this standard work merits the attention of those who are familiar with the language.

C. P. F.

DIE STREPTOKOKKENINFEKTIONEN DES EUTERS INSBESONDERE DER GELBE GALT. (Streptococcic Infections of the Udder with Special Reference to Those Having a Yellowish Discharge.) M. Seelemann, Principal, Institute of Milk Hygiene, Thiel. 259 pages. M. & H. Schaper, Hanover, 1932.

This book gives the results of work carried on in Schleswig-Holstein during the years 1926 to 1931. Mastitis has become a dominent problem in the care of dairy cattle. Just lately in this country investigators are making a careful study of this very common condition. It is undoubtedly true that mastitis is the most common and the most serious disease affecting cattle. Streptococci, of course, constitute one of the most common causes.

This work is divided into eleven parts, which include discussions on the following: Causes of mastitis with special reference to streptococci; the manner and frequency of infection; technic and methods of examinations of milk; bacteriologic descriptions of streptococci infecting the udder; duration of the infection and the treatment and prevention of mastitis. There is appended a list of 219 references to the literature, most of which are from the German. There is included some of the work that has been carried on in this country. However, other work done in America, directly relating to this subject, is not included.

There is very little, if anything, new under means of control or treatment. The book, however, is very interesting and important, especially to those working in this field of milk hygiene and diseases of the udder. We can thoroughly recommend it to those interested in these branches.

C. P. F.

Examining Boards Appointed

The members of the Mississippi State Board of Veterinary Examiners, recently appointed by Governor Conner, began their four-year term of office on June 13, 1932. They are: Drs. Will H. Ewing, Jackson; S. H. Davis, Gulfport; William P. Ferguson (reappointed), Grenada; M. J. Luster, Clarksdale; J. T. Alston, Tupelo.

Governor Pollard of Virginia has appointed the following as members of the State Board of Veterinary Examiners for the ensuing four years: Drs. S. C. Neff, Staunton; H. H. Adair, Bristol; I. D. Wilson, Blacksburg; W. T. Gilchrist, Norfolk; R. E. Ferneyhough, Warrenton.

LE SERO-DIAGNOSTIC DE WRIGHT CHEZ LA BREBIS ATTEINTE DE MELITOCOCCIE DANS LA PERIOD QUI SUIT L'AVORTEMENT (The Agglutination Test of Ewes Affected with Melitensis Infection, following Abortion). Dubois and Sollier. Ann. de l'Inst. Past., xlviii (1932), 3, pp. 372-376.

In a new series of researches, the authors confirm their former conclusions to the effect that the agglutination test of ewes infected with *Brucella melitensis* is variable and inconstant when made during the days following abortion. In fact, the test was constantly negative during the first eight days. The optimum period for taking the blood samples is between the ninth and fifteenth days. And, strangely, blood samples taken during the period of digestion give higher agglutination titres than those collected while the ewes were fasting. The difference between the reactions in animals fasted for fourteen hours and those fed a few hours before the samples were taken was striking. A table showing the variations is included in the report:

Ewes	Reaction	
	After Fasting 14 Hours	4 Hours After Eating
3	0	1:10
5	0	0
16	0	1:20
17	1:30	1:100
18	0	1:20
19	1:10	1:20

The experiment was repeated sixteen days later on the same ewes, with approximately the same general results.

L. A. M.

L'ANESTHESIE EPIDURALE CHEZ LES ANIMAUX DOMESTIQUE (Epidural Anesthesia in Domesticated Animals). Cuille and Chelle. Rev. Gén. de Méd. Vét., xl (1931), 475, p. 393.

The authors give a complete history of rachianesthesia, subarachnoid anesthesia and epidural anesthesia in man and ani-

mals, and review the anatomy of the neural canal and epidural space. The epidural space of animals is much more favorable for the perfusion of injected solutions than that of man, whose neural canal above the cord is interrupted by connective partitions which prevent a regular distribution of the liquid. In animals the epidural space is wide open, with few interruptions from tail to occiput and, therefore, it lends itself nicely to the even flow of any liquid injected into it. Occasional anatomical obstacles, however, may defeat the purpose of the procedure. Perforate intervertebral foramina may allow some of the solution to escape to the outside of the vertebral column, or a longitudinal central partition may prevent the fluid from flowing to both sides of the canal and thus cause only unilateral anesthesia. Rapidity of elimination may cut short the duration of the anesthesia, but this objection can be avoided by adding 5 drops of epinephrin (1:1000) to the anesthetic solution.

Low epidural anesthesia induced by making the injection between the first two caudal vertebrae is an ano-perineo-vulvar anesthesia accompanied by anesthesia of the vagina and rectum. Its indications are tumultuous parturition, rigid cervix, torsion of the uterus, fetal dystocia, inversion of the uterus, retained afterbirth and surgical operations of the tail and pudendum.

High epidural anesthesia is induced by making the injection between the sacrum and the first caudal vertebra. It produces complete paralysis of the posterior extremities and produces an anesthesia of all the region behind the umbilicus.

The authors warn against the use of epidural anesthesia for making explorations of the genital organs for the reason that it causes a dilatation of both the vagina and rectum and thus hinders the palpation of the ovaries and uterus.

In the horse only the low operation should be practiced, as its value is limited to operations performed in the standing position. The injection, therefore, is made between the first two caudal vertebrae, which is about a hand's breadth from the root of the tail. The first caudal vertebra of the horse is fixed to the sacrum and must not be mistaken for the sacrum itself in locating the site for making the injection. Its indications are practically the same as in bovine animals.

In the dog the injection is made in the lumbo-sacral space, not without a certain amount of difficulty. While such an injection induces a good anesthesia, the procedure is less important than in large animals.

L. A. M.

LA PROPHYLAXIE DE L'AVORTEMENT INFECTIEUX PAR LES VACCINATION (The Prophylaxis of Bang's Disease by Vaccination). Zwick. Deut. Tierärzt. Wchnschr. (Nov. 14, 1931), pp. 705-711. (Ext., Rev. Gén. de Méd. Vét., xli (1932), 483, pp. 143-144.)

The author reviews the experiences in Reichsgesundheitsamt in 1911-1915 in the use of killed and live cultures on 123 herds containing 5,136 head which furnished useful facts in the appraisal of vaccination. The figures, having been frequently published, are well known. They show that killed cultures (bacterins) lowered the incidence of abortion from 16.51 per cent to 13.29 per cent; and live cultures from 29.09 per cent to 6.36 per cent, that is to say, 78.2 per cent. It was largely this work that gave prominence to the use of live cultures on a large scale throughout the world. Analogous results were obtained by Stockman, Bang, Schemer and Ehrlich, in Europe, and Theobald Smith and Little, in America. Less favorable results were reported by Jensen, but in 1929 and 1930, Preschold reported the decrease of 30.2 per cent to 3 per cent in the number of abortions occurring in 4,200 animals vaccinated. More recently Schumann, of Breslau, practiced vaccination systematically for three years on 106 herds containing 6,223 head. Here the results were less striking. While abortions were completely eliminated in 46 of the herds after the first vaccination (killed cultures for the pregnant and live cultures for the non-pregnant), the total abortions among the 6,223 animals actually showed an increase during the three-year period because the prophylactic vaccination and other measures were not practiced regularly. This example shows the importance of systematically organizing the prophylactic measures, as was done by the abortion commission of Germany, when it formulated the new directions for the control of contagious abortion last year. The author's conclusions are summarized as follows:

1. In the absence of better methods of intervention, live-culture vaccination cannot be prudently dispensed with in Germany in the treatment of badly infected herds.

2. The danger to man from the use of live-culture vaccines is not consequential.

3. Hygienic measures alone are recommended for herds only slightly affected and it should be insisted upon that these be practiced more generally by farmers.

4. As recommended in the resolutions passed at the Eleventh International Veterinary Congress at London and the Office In-

ternational des Epizootics, efforts should be made to discover a prophylactic measure that could replace living vaccines.

L. A. M.

UNDERSÖKNING ÖVER FÖREKOMST AV B. ABORTUS BANG I MJÖLK FRÅN KOR, SOM KASTAT TILL FÖLJD AV INFEKTION MED DENNA BAKTERIE (Investigations Concerning the Occurrence of *Bact. abortus* Bang in Milk from Cows Which Have Aborted in Consequence of Infection with This Bacterium). E. Klarin. Skand. Vet.-Tids., xxi (1931), pp. 249-269.

This investigation consisted of a study of 60 cows and heifers that had aborted as the result of Bang's disease. In 57 instances the diagnosis was made by a microscopic examination of the placenta. In the remaining three cows it was assumed that the abortion was the result of Bang's disease, as both the agglutination blood-tests and milk samples were positive at the time of the first samples, which was 7 days following the abortion. The 60 animals were from 37 herds.

A definite amount of milk was taken from each teat, in all 250 cc. The samples were taken in sterile glass vessels and at such a time that they could be shipped to the laboratory and used the same day or, at the latest, the next day. No preservative was used and only during the summer was it necessary in a few instances to obtain a second sample because of the first becoming sour in transit. As soon as the milk arrived at the laboratory, it was centrifuged for 15 minutes at 3,500 r.p.m. The cream layer became of salve consistency. About 7 cc of this cream was taken up in a metal measure. For each test 2 guinea pigs were injected with 1 cc of cream. At first the injections were made intramuscularly, later subcutaneously. The guinea pigs were killed. Blood samples were taken at four weeks and six weeks following injection. Samples were taken from the cows at regular intervals: 7, 14, 21, 28, 48, 56, 90, 120 and 150 days.

The first studies concerned the value of cream and sediment as materials to be injected. Two groups of animals were used. One group of 16 animals was injected with cream and sediment and the guinea pigs killed four weeks after injection. The other group of 24 animals was treated on the same day, but the guinea pigs were killed six weeks after injection. The conclusions are that it is best to use both the cream and sediment and if only one of these is to be used, then cream is a little more certain, although it is not entirely satisfactory.

In the second study of 8 samples of milk, half of the guinea

pigs were injected with 1 cc of cream, the remainder receiving 0.5 cc. Results were that it is more suitable to use 0.5 cc.

Another study embraced a comparison of 1 cc and 0.1 cc injections, with four- and six-week intervals between the injection and the killing of the guinea pigs. In all, 96 samples of milk were tested: 34 at the four-week interval and 62 at the six-week interval. The conclusion was that 0.1 cc is too uncertain, compared with 1 cc.

A comparison test was made between 0.5 cc and 0.1 cc of cream on 11 samples. The conclusion was that 0.5 cc of cream was more suitable.

In two groups of 32 guinea pigs, one month after injection, the mortality in the 1-cc and 0.5-cc series was respectively 10 and 3.

A comparative study was made on the interval between the injection and the bleeding of the guinea pig, one series having a four-week interval and the other a six-week interval, with the following results: The dosage was 1 cc of cream. Higher agglutination titres at six weeks than at four weeks in 176 cases (71 per cent). The same agglutination titres at six weeks as at four weeks in 32 cases (13 per cent). Lower agglutination titres at six weeks than at four weeks in 41 cases (16 per cent). This was repeated with 0.1-cc dose with similar results, *i. e.*, the six-week interval between injection and bleeding was more satisfactory.

A study was made between intraperitoneal injection with 1 cc at a three-week interval and subcutaneous at four weeks; 42 tests from 26 cows gave the following results: higher aggutination titres in the intraperitoneally injected pigs in 6 cases, same agglutination titres in the intraperitoneally injected pigs as in subcutaneous injections in 2 cases, and lower agglutination titres in the intraperitoneally injected pigs in 34 cases.

Therefore, it seems that the pigs subcutaneously injected at 4-week intervals are the most desirable. During the study of these 60 cows it was found that 52 (87 per cent) eliminated *Bact. abortus* Bang in the milk at some time.

The author divides the positive cows into two groups:

Group I includes 33 cows which were positive 7 days following abortion, and 10 cows which were positive within the first month following abortion. Therefore, 43 cows (70 per cent) eliminated the Bang germ in the milk in this group. In 25 cases the organism was eliminated 2 months or more following abortion. One cow was included in group I which was studied for less than 2 months. In the remaining 17 cows the elimination was

of only short duration. Also a reinfection took place in 9 cows at a later date.

Group II includes those animals which begin to eliminate *Bact. abortus* in the milk at such a late period after abortion that it cannot be looked upon as the result of autogenous infection from the uterus. In this group are classed 9 animals which first became positive 6 weeks following abortion. In these cases only an occasional sample has been positive. As previously mentioned, in this group are placed 9 individuals that were reinfected; therefore, 18 cows (30 per cent) are positive, not as the result of infection at the time of abortion, but perhaps from the surroundings. Eight cows (13 per cent) were negative throughout the experiment.

W. L. N.

AKUT DIFFUS GLOMERULONEPHRITIS HOS SVIN (Acute Diffuse Glomerulonephritis in Swine). J. Egehøj. Maan. f. Dyrl., xliii (1931), pp. 225-235.

One often finds, during an examination of a decapsulated kidney from slaughtered swine, many small hemorrhages. A histological examination of 187 such kidneys disclosed three examples which should be classified as acute diffuse glomerulonephritis.

Glomerulonephritis may be designated as diffuse or localized, acute, subacute, or chronic. It is a debatable question whether a primary localized glomerulonephritis can exist when found in conjunction with the embolic interstitial type, since in the latter one finds changes in the Malpighian corpuscles which closely approximate an embolic process. The histological changes noted in the glomeruli and Bowman's capsules, in the instances here reported, are on the whole identical with those found in human cases in acute diffuse glomerulonephritis. Most authors, however, designate the condition as embolic nephritis.

Acute diffuse glomerulonephritis may be subdivided further into the intra- and extracapillary forms. The former is characterized by a primary degeneration of the Malpighian corpuscles, while in the latter the most definite lesions are distinct from the glomerulus, namely, a proliferated process in the epithelium of Bowman's capsule.

These diseases have been investigated to only a small extent in farm animals. Neiberle described cases of diffuse glomerulonephritis in cattle and swine. He classified under diffuse glomerulonephitis the well-known types of large, light kidneys in cattle which were formerly considered as examples of chronic

parenchymatous nephritis. Primgaard, however, disagrees with Nieberle's conclusion, and after extensive study states that the condition is an amyloid degeneration. F. Henschen believes that it would be impossible to distinguish the diffuse glomerulonephritis in swine when dissociated from the so-called hemorrhagic glomerulonephritis accompanying swine erysipelas. According to this latter author, the nephritis found in swine erysipelas can scarcely be classified as glomerulonephritis comparable to that in human beings. Except for these citations the literature contains but few references to cases of glomerulonephritis in domestic animals.

S. Larson has recently contributed an important article on the kidney diseases of swine which he attributes to the feeding of spoiled fodder. The pathological changes which he described were found in the Malpighian corpuscles but were of a different nature from typical diffuse glomerulonephritis. This author also lays particular emphasis on simultaneously occurring changes in the tubular epithelium.

Microscopic changes found in the three kidneys mentioned briefly are as follows: The glomeruli were enlarged to the extent that they filled Bowman's capsule. Low magnification showed a homogeneous mass surrounding the glomerulus. Higher power showed that the capillaries in the glomerulus are free of blood. Some sections show an increase in the number of cell nuclei in the glomerulus, others show a very decreased number. Whenever an increased number of nuclei are present, one finds both lymphocytes and leukocytes in addition to the usual vessel, endothelial cells. In all three specimens, individual glomeruli were noted which contained cells, the protoplasm of which had acid, basic or neutrophilic granules. The acidophilic granulocytes, however, were in the majority. In one case 265 granulocytes were noted in 10 glomeruli. Changes were apparent also in the connective tissue structure of the kidney.

Many sections of the tubules showed an exudate present which caused an enlargement of the lumen. Some sections showed an increase in the perivascular tissue resulting from cell proliferation and infiltration. A careful comparison of the lesions found in the three specimens indicates that the lesions are a combination of glomerulo- and embolic-interstitial nephritis, with the glomerular changes of the greater importance.

The cause of glomerulonephritis may be either infection or intoxication. In embolic nephritis the mode of infection is of primary consideration. It is possible that an infection which produces glomerulonephritis is so extensive, namely, it forms so large a supply of infective material, that it simultaneously

gives rise to a reaction from the vascular portions of the kidney. Such a condition may be the cause of the three cases here described.

When one considers the extent and severity of the lesions of the kidneys here described, it is peculiar that the antemortem observations do not indicate disease. The pigs from which these diseased kidneys originated were without symptoms of any known disease. After slaughter, no pathological lesions were found except those of the kidneys and their accompanying lesions. The carcasses of these swine were well conditioned and without urinary odor. Of course it is true that in man glomerulonephritis may exist without any observable symptoms, the condition oftentimes remaining unknown until autopsy reveals the lesions of glomerulonephritis.

W. L. N.

BETRACHTUNG ÜBER BANG-LEBENDKULTER,-VAKZINE, FARB-IMPFSTOFF (RAKULIN) (Observations Concerning Bang-Living Culture,-Vaccine, Colored-Vaccine [Rakulin]). G. Berthold. Berl. Tierärzt. Wchnschr., xlviii (1932), 16.

A vaccine for the control of Bang's disease has been devised which is called, in this article, "Rakulin's Bang colored vaccine." The author states that the protein has been set free from the organism so that it functions immediately as an antigen. The advantages claimed for this product are : (1) its rapid resorption, (2) sterility, (3) lack of local reaction and (4) little, if any, after-effects.

M. D. K.

UEBER DIE EMPFINDLICHKET DER KATZEN GEGEN BRUCELLA MELITENSIS-ABORTUS (Concerning the Sensitiveness of Cats to *Brucella Melitensis-Abortus*). W. N. Makkawejsky and I. A. Karkadinowskaja. Deut. Tierärzt. Wchnschr., xl (1932), 15.

Bact. abortus Bang is pathogenic for a large group of animals. The latest additions to this group showing sensitiveness to this germ are birds and horses. There is little information in the literature concerning the sensitiveness of cats for the Brucella group. Shaw reports that he tested 22 cats for melitensis on the island of Malta, five of which gave positive agglutination tests. Positive cases have been described also in Frankreich. The author injected 16 cats with a suspension of Brucella. The types of injection were subcutaneous and intraperitoneal. Eight were injected with the abortus type and eight with the melitensis type. A separate strain was used for each animal. The dose

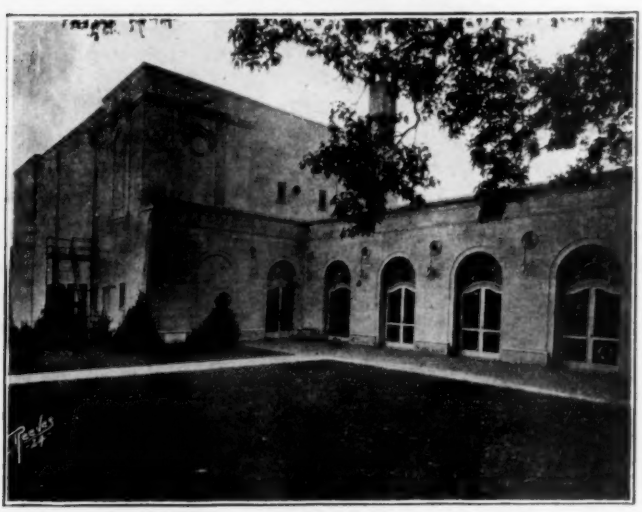
for the inoculations was 0.1 cc of the suspension. The injected cats were placed in separate cages. During the course of observation, which extended 187 days, only five showed clinical evidence of disease; three died; the remaining 13, after various lengths of time, were destroyed and carefully examined and cultured. The author concludes that the Brucella group is pathogenic by the subcutaneous and intraperitoneal methods of injection for cats. The clinical changes shown were weakness, loss of appetite, emaciation, conjunctivitis, cough, and swelling and painfulness of the joints. In the majority of cases, however, the symptoms of the disease were not observed. Three of the inoculated animals died in 10 to 75 days. Agglutinins appeared in the blood in dilutions of from 1:500 to 1:3200. This was determined in six animals. In five cases the organism was isolated after death. The majority of the cases of cats destroyed in 125 to 187 days were negative upon bacteriologic examination.

M. D. K.

Foot-and-Mouth Disease in England

During the twelve months ended April 23, 1932, a total of 3,654 cattle, 5,409 sheep, and 1,204 hogs were slaughtered in England for foot-and-mouth disease. The owners were paid £95,483 in compensation.

ON TO ATLANTA



The Atlanta Women's Club, a beautiful building, wonderfully furnished. It is occupied exclusively by the club.

Regular Army

Major John H. Kintner is relieved from further assignment at Randolph Field, Tex., and will proceed to Fort Sam Houston, Tex., for duty.

Major Fred C. Waters, in addition to his other duties at Seattle, Wash., will report to the commanding officer, Fort Lawton, Wash., for duty.

Captain Edwin K. Rogers has been directed to proceed to his home to await retirement.

The promotion of Captain Raymond T. Seymour and Captain Oscar C. Schwalm to the grade of Major, to rank from June 17 and 18, respectively, is announced.

Veterinary Reserve Corps

New Acceptances

Berry, Dalys Lewis....2nd Lt....Wilsey, Kan.
Boley, Loyd Edwin....2nd Lt....R. F. D. 2, Topeka, Kan.
Clark, Virgil Howard..2nd Lt....R. F. D. 1, Webber, Kan.
Dean, Ben Harrison...2nd Lt....717 Humboldt St., Manhattan, Kan.
Dimon, Charles E.....2nd Lt....219 N. Juliette St., Manhattan, Kan.
George, John Lester...2nd Lt....Chester, Neb.
Hartzell, Harold P....2nd Lt....1221 Thurston St., Manhattan, Kan.
Hodgson, Melvin E....2nd Lt....R. F. D. 1, Hutchinson, Kan.
Hornsby, Will S., Jr..2nd Lt....Millington, Tenn.
Paige, Chester Anson..2nd Lt....219 N. Juliette St., Manhattan, Kan.
Patton, Glen Frank....2nd Lt....R. F. D. 3, Cawker City, Kan.
Van Meveren, A. F....2nd Lt....Orange City, Iowa.

Promotion

Broad, Fay Elton....To Capt....802 N. Michigan, Plymouth, Ind.

Transferred

Walker, Erling H.....2nd Lt....Auburn, Ala., from FA-Res.

Consumption of Rabbit Meat in England

Annual sales of 1,000 to 1,200 tons of domestic rabbit carcasses at Smithfield Market, London, Eng., indicate the importance of the rabbit as a meat product. In addition, there were importations into England during 1931 of fresh rabbits valued at £196,649; frozen rabbits valued at £519,408, and canned rabbits valued at £13,103.

MISCELLANEOUS

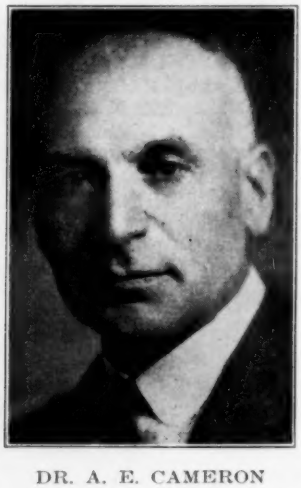
Introducing Doctor Cameron

In the July issue of the JOURNAL it was announced that Dr. A. E. Cameron, of Ottawa, Ont., had been elected a member of the Executive Board of the A. V. M. A. to represent Canada (District 1). Since this announcement was made we have gathered together a few interesting facts concerning Dr. Cameron.

By birth a Scotchman, born near Dundee, at Brought Ferry, in 1878.

Attended high school and the Morgan Academy at Dundee.

Migrated to London, did not like the big city, and went to South Africa.



DR. A. E. CAMERON

Joined the Rhodesian Mounted Police and later took an active part in the Boer War. Demobilized in 1900.

Became sailor and boarded a four-masted bark for Australia. Accompanied shipment of horses to Singapore. Went to the Philippines and thence to Hong Kong.

Returned to London (1903) and sailed for Montreal (1904). Homesteaded in Saskatchewan. Still owns farm near Regina.

Entered Ontario Veterinary College (1906) and was graduated (1908) with honors. Medalist in physiology and anatomy and prize-winner in all other subjects.

Practiced a few months in Saskatchewan and then entered service of federal Department of Agriculture. Assigned to meat inspection at Winnipeg.

Transferred to field work in Saskatchewan (1911). Entered laboratory at Lethbridge (1914).

In military service again, from May, 1915, until July, 1919.

Returned to Canada and proceeded to Alberta to take charge of field work for Health of Animals Branch. Took charge of Veterinary Research Station at Lethbridge (1920).

Appointed Chief Veterinary Inspector (1925). As such, is assistant to Veterinary Director General.

Commissioned lieutenant-colonel in Canadian Army Veterinary Corps. Holds Military Cross and auxiliary officers' long service decoration of V. D. Vice-president of Professional Institute of the Civil Service of Canada.

Member of Saskatchewan Veterinary Association, Central Canada Veterinary Association, Canadian Public Health Association and American Society of Parasitology.

Texas Ladies' Auxiliary Meets

The Ladies' Auxiliary to the State Veterinary Medical Association of Texas met concurrently with that organization at College Station, June 6-10, 1932, with 26 members in attendance. Auxiliary headquarters were at Aggieland Inn.

Various activities were scheduled for the entertainment of the members, including a visit to the historic home of the late Elizabeth Ney, the sculptress, and her husband, the late Dr. Robert Montgomery, noted physician and philosopher, and a tour of inspection of the State Normal and Industrial College for Negroes at Prairie View.

Among the hostesses who entertained the ladies at luncheons and other functions during their stay were Mesdames R. P. Marsteller, George Long, P. W. Burns, A. A. Lenert, A. E. Wharton, Dee Pearce and U. E. Marney.

At the business session of the Auxiliary meeting, nine new members were received, and the following officers were elected: President, Mrs. L. I. Lucy, Wichita Falls; first vice-president, Mrs. Mark Francis, College Station; second vice-president, Mrs. C. W. Neal, San Antonio; secretary-treasurer, Mrs. U. E. Marney, San Antonio (reëlected). The next meeting is to be held at San Antonio.

KANSAS CITY VETERINARY ASSOCIATION

The Kansas City Veterinary Association recently was incorporated under the laws of Missouri. Previous to this action, the enabling revision of the constitution and by-laws was made. The Association now meets monthly, on the third Tuesday evening of each month.

The following officers have been elected to serve during the ensuing year: President, Dr. H. L. Bussong, Belton; vice-president, Dr. F. L. Seevers, Pleasant Hill; secretary-treasurer, Dr. J. D. Ray, Kansas City (reëlected).

The Association has had good attendance at all recent meetings and a record attendance of 90 veterinarians at a special meeting held April 19.

At the meeting held on May 17, a program pertaining to small animals was presented. Dr. J. C. Flynn was in charge of this program. Dr. Calvin Cooper, Director of the Health Department of Kansas City, was present and discussed the rabies situation in Kansas City, and the relation of the practicing veterinarians to the Health Department, in the matter of immunizing dogs against rabies.

Dr. A. R. McLaughlin presented a very interesting paper on some pathological conditions of the blood, with special reference to chlorids of the blood. Dr. H. W. Young talked on the subject of "Intestinal Disturbances of Small Animals." All of these subjects were well discussed.

The June meeting was held June 21, at the Baltimore Hotel. Dr. S. L. Stewart, of Olathe, Kan., talked on "Applied Anatomy of the Digestive Tract of Horses." This was a very instructive talk and a good discussion followed. Several interesting case reports were given and a round table on heat prostration in horses was conducted by Dr. H. L. Bussong.

It was decided to dispense with the July meeting, in deference to the Missouri Veterinary Medical Association meeting at Springfield, July 20 and 21.

J. D. RAY, *Secretary.*

SOUTHWESTERN MISSOURI VETERINARY MEDICAL ASSOCIATION

The Southwestern Missouri Veterinary Medical Association held its spring meeting at Monett, Mo., May 25, 1932. Dr. R. F. Whitlock, of Monett, made the local arrangements and the meeting was held in the beautiful city park. The band-stand in the grove was used for the occasion.

The program was begun at 10 a. m. Dr. C. D. Meredith, of Joplin, presented the subject of "Swine Erysipelas and Differential Diagnosis." Dr. H. J. MacCartney, of Joplin, chose as his subject, "Acetonemia of Cattle." A good discussion followed each of these subjects.

Dinner was served at the clubhouse in the park. The wives of the veterinarians and several local business men were guests at the dinner. Following this, Dr. A. M. McCapes, of the Extension Department of the University of Missouri, talked on the coöperation of the state authorities with the local veterinarians, in Bang's disease control and bot eradication. This was a very interesting subject to all present, and an active discussion followed.

Dr. C. D. Folse, of Kansas City, conducted a question-box which brought forth much good information and many timely subjects and case reports.

The following officers were elected for the coming year: President, Dr. C. D. Meredith, Joplin; vice-president, Dr. W. J. Stone, Joplin; secretary-treasurer, Dr. R. F. Whitlock, Monett.

After adjournment of the regular meeting, several pigs were presented for autopsy and clinic. Dr. Ashe Lockhart, of Kansas City, discussed the postmortem findings.

J. D. RAY, *Reporter.*

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The twenty-second annual meeting of the State Veterinary Medical Association of Texas and the tenth Short Course for Graduate Veterinarians opened in Francis Hall, Texas A. & M. College, June 7, 1932, with 62 members and visitors in attendance. Dr. Mark Francis gave his annual address, welcoming the men with all his usual fire and vigor. He gave a detailed account of the new veterinary hospital soon to be under construction, of which the College is justly proud, as it is to be one of the finest and best equipped in the State.

Dr. R. P. Marsteller introduced Dr. W. F. Guard, of Ohio State

University, and Dr. Frank Breed, of Norden Laboratories, Lincoln, Neb. Those in attendance appreciated Drs. Guard and Breed very much. Dr. Guard's work was thorough and he was called upon often. The "Cowboy of Texas" has been heard of often. Dr. Guard's subject was the cow, and before the Short Course was over it was suspected that Ohio had a cowboy with knife and medicine.

Dr. J. K. Northway, veterinarian at King's Ranch and manager of Santa Gertrude's Jersey Farm, Kingsville, assisted by Dr. Verne A. Scott, of John Tarleton College, Stephensville, gave a demonstration on the use of the cowboy's rope and its help in spaying heifers. Dr. Northway spays about 300 a day. He also gave a lecture on what was being done to improve the beef types of cattle on the ranches. The eastern Texas men enjoyed the western methods and demonstrations very much.

A chicken barbecue, with many other good eats, was tendered the members and vistors, and was thoroughly enjoyed by the 75 who were present. Afterwards a delightful social hour was had with the ladies of the Auxiliary, who had assisted with the banquet.

A business session followed, after which there was a round-table of heart-to-heart talks. Calcium chlorid was a favorite number, some veterinarians making calcium talks, and sometimes calcium making the veterinarians talk. Dr. Breed stated that the proper administration of a dose should take thirty minutes.

An extensive program on small animals was carried out, with Dr. Fred A. Murray, Austin, second vice-president, presiding:

Mineral Requirements for Dogs—Dr. P. W. Burns, A. & M. College of Texas.

Control and Eradication of Ticks and Fleas in Kennels and on Private Premises—Dr. U. E. Marney, San Antonio.

Canine Distemper—Dr. R. A. Self, Dallas.

The following practitioners, assisted by Dr. A. A. Lenert, A. & M. College of Texas, conducted a small-animal clinic: Drs. W. G. Brock, Dallas; L. E. Casey, Dallas; R. A. Self, Dallas, and W. R. McCuistion, Fort Worth.

In connection with the meeting the Board of Veterinary Medical Examiners admitted to practice six recent graduates: Drs. M. N. Bader, Wharton; Fred W. Pease, Austin; R. A. Goodman, Bryan; Elmer A. Maier, Fredericksburg; Samuel E. Grove, Austin, and John M. Fitte, New Orleans, La.

The following officers were elected for the ensuing year: President, Dr. W. F. Hayes, Farmersville; first vice-president, Dr. Fred A. Murray, Austin; second vice-president, Dr. James

K. Northway, Kingsville; secretary-treasurer, Dr. D. Pearce, Leonard (reëlected).

D. PEARCE, *Secretary.*

CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The twenty-second annual meeting of the Central New York Veterinary Medical Association was held at the Cantie Chiel Club, at Canastota, N. Y., June 11, 1932. The meeting opened at 3 p. m., with the President, Dr. D. M. Hoyt, in the chair. Roll-call by the Secretary showed 25 members and two visitors present.

The following officers were elected for the coming year: President, Dr. W. F. Burleigh, Oriskany Falls; vice-president, Dr. W. M. Sullivan, Marcellus; secretary-treasurer, Dr. W. B. Switzer, Oswego (reëlected). The present censors were reëlected.

It was then decided to adjourn for a clinic, at which Dr. C. H. Case, of Akron, Ohio, gave a very instructive talk and demonstration on sterility and abortion, after which we returned to the club, where Dr. S. D. Johnson, of the New York State Veterinary College, gave a very interesting demonstration of the test for mastitis. This was followed by a discussion.

Dr. W. H. Bales, Oswego County Veterinarian, discussed county-area eradication of tuberculosis. Discussion followed.

It was moved, seconded and carried that the chair appoint a Grievance Committee of three to confer with Dr. J. C. Stevens and give him every assistance available. Drs. W. I. Clark, J. B. Knapp and W. F. Burleigh were appointed as such committee.

We then adjourned to meet with the ladies, at Schrays, at Chittenango, for dinner. The ladies met at the residence of Dr. Hoyt at 2 p. m., and were taken by Mrs. Hoyt for a very pleasant drive through Chittenango Falls, Cazenovia, Cazenovia Lake and back to Chittenango for dinner at 6 p. m. We broke up at 10 p. m., after a very profitable and enjoyable meeting.

W. B. SWITZER, *Secretary.*

EAST TENNESSEE VETERINARY MEDICAL SOCIETY

The East Tennessee Veterinary Medical Society met at the hospital of Drs. Morgan and Hatchett, in Chattanooga, June 11, 1932. Plans for the A. V. M. A. convention in Atlanta were discussed by Drs. F. W. Morgan and M. Jacob. Case reports were made by Drs. Jones, Logan, Hatchett, Adair, Kagey and Bauch, followed by a clinic.

R. E. BAKER, *Secretary.*

SOUTHEAST KANSAS VETERINARY MEDICAL SOCIETY

The Southeast Kansas Veterinary Medical Society held a very enjoyable meeting at Fort Scott, June 16, 1932. The meeting, which consisted of a clinical program followed by a basket lunch, was the first of its kind held by the society, and was attended by seventeen Kansas veterinarians and four from Missouri.

The clinic was held in Dr. Richardson's veterinary hospital. Drs. Dykstra, Frick and Lumb, of the Veterinary Division of the Kansas State College, Manhattan, were in charge of the program, which was as follows:

Dr. Dykstra demonstrated and discussed the tail operations of the horse, removal of the cancerous membrana nictitans, and actinomycosis in cattle. Dr. Frick demonstrated the spaying operation, the use of nembutal and epidural anesthesia, and the treatment of parasitic mange and eczema in small animals. Dr. Lumb discussed poultry diseases and supplemented his discussions with autopsies. Numerous other autopsies were held and discussed by the veterinarians present.

Following the clinical program, a basket lunch was served in the city park by the wives of the veterinarians in attendance.

The September meeting of the society will be held at Independence.

L. F. BARTHELME, *Secretary.*

NORTH CAROLINA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-first annual meeting of the North Carolina State Veterinary Medical Association, held at Henderson, N. C., June 29-30, 1932, was a joint meeting with the Virginia State Veterinary Medical Association. Approximately 100 veterinarians were present from the two states. This is practically a 50 per cent attendance of all the veterinarians from the two states, and 70 per cent of the membership of the two associations. About 50 ladies were in attendance and enjoyed a full program of activities.

Dr. P. C. McLain, president of the North Carolina Association, presided the first day, and Dr. W. H. Grubb, the Virginia president, was in charge the second day. The morning of each day was taken up with the presentation of papers. Dr. L. E. Starr, of the Virginia Polytechnic Institute, gave an excellent paper on "Undulant Fever." Dr. John H. Hamilton, of Raleigh, with the North Carolina State Board of Health, read an interesting paper

on "Public Health Significance of Diseases Primarily of Animals, Secondarily of Man." Dr. R. R. Dykstra, president of the A.V.M.A., made a very practical talk on the use of anesthetics, stressing the use to be made of chloral hydrate as a hypnotic. He recommended using from one to two ounces of chloral hydrate, dissolved in one to two pints of normal saline solution and given to horses intravenously. Small horses receive one pint and large horses two pints. One-quarter grain atropin sulfate, given subcutaneously previous to the intravenous injection of the chloral hydrate solution, will largely prevent the depressing action on the heart. From 10 to 15 cc of camphorated oil is an antidote for chloral hydrate poisoning. On the second afternoon Dr. Dykstra conducted a large-animal clinic.

A symposium on meat and milk inspection was held on the second morning. Papers were presented by Dr. H. C. Givens, State Veterinarian of Virginia; Major V. B. Wright, Veterinary Corps, U. S. Army, Fort Bragg, N. C., and Dr. U. G. Houck, Associate Chief, Bureau of Animal Industry, Washington, D. C. This subject was discussed by Dr. C. A. Cary, of Auburn, Ala. Professor W. L. Threlkeld, of the Virginia Polytechnic Institute, read a valuable paper on "The Internal Parasites of Sheep." The parasites of dogs were discussed in a splendid talk by Dr. Walter R. Hobbs, of Ohio State University. He brought out the interesting fact that certain forms of tapeworms of dogs may be more harmful to man than to the dog. Arecolin gives variable results in expelling tapeworms. Its action is materially assisted by high enemas of normal saline solution. The first afternoon, Dr. Hobbs conducted the small-animal clinic. At this clinic Dr. F. D. McCrea, of Duke University, Durham, N. C., performed a debarking operation on a dog.

Dr. H. C. Givens was the toastmaster at the banquet, Wednesday evening, which was featured by Dr. I. D. Wilson, secretary of the Virginia Association, giving a paper on "The Destiny of Mankind." At the business session which followed, the following officers were elected: President, L. J. Faulhaber, Raleigh; 1st vice-president, C. L. Cruse, Salisbury; 2nd vice-president, T. L. Glenn, Mocksville; secretary-treasurer, J. Howard Brown, Rich Square (re-elected).

One item of business of more than local interest was the decision to affiliate with the A.V.M.A. under the proposed plan submitted by the Committee on Affiliation. Hendersonville, N. C., was selected as the 1933 meeting place.

J. HOWARD BROWN, *Secretary.*

NECROLOGY

THOMAS A. NEWELL

Dr. Thomas A. Newell, prominent Chicago veterinarian, died suddenly from a heart attack in December, 1931, while mounting the stairway of the elevated railway station at the Union Stock Yards, where he was engaged temporarily as an inspector for the State Department of Agriculture.

In his younger days, Dr. Newell was an outstanding coachman and whip among the best families of Chicago and upon his graduation from the McKillip Veterinary College in 1900 he became the trusted veterinarian of the same circle. His clients were the Armours, the Meekers, the Pullmans, the Lowdens and, with the passing of the coach horse, his services went to the business connections of the same circle.

Few men in the veterinary profession can boast of having the confidence of their clients to the same extent as did Tom Newell, as he was affectionately called by his friends. He was the veterinarian of ex-Governor Lowden's famous Holstein-Friesian farm at Oregon, Illinois, until the time of his sudden death. He is survived by his widow and a son. L. A. M.

DAVID McAUSLIN

Dr. David McAuslin, of Brooklyn, N. Y., died March 10, 1932, after a brief illness. He was born in London, England, December 18, 1880, and was a graduate of the New York-American Veterinary College, class of 1907. With his brother, the late Dr. Robert A. McAuslin (N. Y.-Amer. '02) he conducted the Brooklyn Veterinary Hospital, at 397 Carlton Ave. A sister survives him.

Dr. McAuslin joined the A. V. M. A. in 1918. He was a member of the Brooklyn Riding and Driving Club and the Saddle River Polo Club. Prior to the entry of the United States in the Great War, Dr. McAuslin was employed by the Remount Division of the Italian Army. Later he served as a first lieutenant in the Veterinary Corps of the U. S. Army. He also held a com-

mission as captain in the 105th Cavalry, New York National Guard.

THOMAS FARMER

Dr. Thomas Farmer, of Grand Blanc, Mich., the oldest graduate veterinarian in Michigan, died at his home, May 26, 1932, following an illness of six months.

Born at Montgomeryshire, North Wales, November 1, 1845, Dr. Farmer came to America at the age of four years. The family located at Perth, Ontario, where Dr. Farmer remained until 1866. That year he located at Hadley, Mich., where he followed his trade as blacksmith and operated a stage coach between Hadley and Grand Blanc. He removed to the latter place in 1875. Later, though he had a family of five children to rear and educate, he decided to study veterinary medicine. He went to Chicago and entered the Chicago Veterinary College. Following his graduation in 1888, he returned to Grand Blanc and practiced his profession there for 44 years.

Dr. Farmer joined the A. V. M. A. in 1908. After many years of active membership in the Michigan State Veterinary Medical Association, he was placed on the roll of honorary members. For nine years he served as a member of the Michigan State Board of Veterinary Medical Examiners. He was president of the local school board for 35 years, and took a prominent part in organizing the local consolidated school, the first of its kind in Michigan.

Surviving Dr. Farmer are four daughters, one son, two brothers, two half-brothers and two sisters.

SAMUEL E. SPRINGER

Dr. Samuel E. Springer, of Casper, Wyo., died in Denver, Colo., June 20, 1932, a few hours following an operation for the removal of a goiter. He was a graduate of Ohio State University, class of 1904, and entered the service of the U. S. Bureau of Animal Industry soon thereafter. He was assigned, at various times, to meat inspection at Omaha, Neb., tick eradication in Texas and scabies eradication in Colorado. For the past 25 years he had been employed in field work in Colorado and Wyoming. He served for a few weeks in California during the recent outbreak of foot-and-mouth disease.

Dr. Springer joined the A. V. M. A. in 1910. He was a Mason and held membership in the Blue Lodge, Consistory and Shrine. He is survived by two brothers. Burial was at Fairview, Ohio.

C. E. W.

B. F. KINYON

Dr. B. F. Kinyon, of Ladysmith, Wis., died at his home, May 26, 1932. He was born at Sharon, Wis., February 19, 1856, and was a graduate of the Chicago Veterinary College, class of 1891. He practiced at Clinton, Wis., until 1908, when he moved to Ladysmith. He joined the A. V. M. A. in 1911. Surviving him are his widow, two daughters and one son. Funeral services were under Masonic auspices.

JOSEPH W. UPRIGHT

Dr. J. W. Upright, of Oshkosh, Wis., died at Mercy Hospital, that city, June 25, 1932, after a brief illness. Born at Little Falls, N. Y., July 11, 1886, he studied veterinary medicine at the Ontario Veterinary College. He was graduated in 1911 and practiced for some time in Denmark, Wis. He had lived in Oshkosh for 11 years, during the past five of which he had been in the employ of the State Department of Agriculture. Dr. Upright was a member of the Wisconsin Veterinary Medical Association. He is survived by his widow, four sons, his step-father and mother, two brothers and two half-sisters. The body was taken to Kenosha for burial.

ADAM F. ELLIOTT

Dr. Adam F. Elliott, of Milton, N. Dak., died in a Minneapolis hospital, June 25, 1932, after a long illness. He was a graduate of the Ontario Veterinary College, class of 1896, and had been in practice at Grandin and Milton, N. Dak., for 35 years.

Dr. Elliott joined the A. V. M. A. in 1912. He is survived by his widow, one daughter and two brothers.

THOMAS A. ATTRIDGE

Dr. Thomas A. Attridge, of Harbor Beach, Mich., died at his home, June 30, 1932, after a long illness. Born December 4, 1865, in Highgate, Ontario, he studied veterinary medicine in the Veterinary Department of the Detroit College of Medicine. Following his graduation in 1894, he located at Harbor Beach and remained there until his death. He was a member of the Michigan State Veterinary Medical Association, and for many years served as County Live Stock Agent. He is survived by his widow, one daughter, one son, four brothers and two sisters.

ALEXANDER LOWE

Dr. Alexander Lowe, aged 63, who had practiced as a veterinarian at Philadelphia, Ind., for 30 years, was fatally injured when he suffered a broken vertebra in a crossroads automobile accident on June 19, 1932. He lived for 11 days at St. Vincent's Hospital, Indianapolis, dying on June 30. He is survived by his widow (née Flora Fry), one daughter, three brothers, one of whom is Dr. O. E. Lowe (Ind. '12), of Charlottesville, Ind., and five sisters. Dr. O. B. Curry (O. S. U. '25), of Morristown, Ind., is a nephew of the deceased.

CHARLES H. DECKER

Dr. Charles H. Decker, of Columbus, Ohio, died at the home of his mother, in Columbus, July 18, 1932. Death followed a sinus operation.

Born at Martel, Ohio, January 23, 1890, Dr. Decker attended grade and high schools before entering Ohio State University. He finished the course in veterinary medicine in 1910. For a number of years he was in the service of the U. S. Bureau of Animal Industry and was engaged in tuberculosis eradication work in Ohio.

Dr. Decker joined the A. V. M. A. in 1929. He was also a member of the Ohio State Veterinary Medical Association. He is survived by his widow (née Pauline Blackford), his mother, two sisters and one brother. Interment was at Iberia, Ohio.

JEROME ORR

Dr. Jerome Orr, of Fort Lauderdale, Fla., died July 19, 1932. Born near Uniopolis, Ohio, September 12, 1869, Dr. Orr studied veterinary medicine at the Ontario Veterinary College. Following his graduation in 1907, he located at Wapakoneta, Ohio, later going to Nahunta, Ga. Five years ago he removed to Fort Lauderdale, Fla. Funeral services and interment took place at Waycross, Ga. Dr. Orr is survived by his widow (née Ida Sibert) and one daughter.

Our sympathy goes out to Dr. S. A. Johnson, of Ithaca, N. Y., in the death of his wife, March 22, 1932, following an operation for appendicitis; to Dr. H. J. Hancock, of Portland, Ore., in the death of his wife, Iona M. Hancock, on May 20, 1932, from cerebral hemorrhage; and to Dr. and Mrs. William Caslick, of Gouverneur, N. Y., in the death of their infant daughter, July 13, 1932.

PERSONALS

MARRIAGES

Dr. C. H. Milks (Corn. '30) to Miss Edith Betty O'Daniel, both of Ithaca, N. Y., July 5, 1932, at Ithaca, N. Y.

Dr. Thomas A. Sigler (Ind. '02) to Miss Florence H. Hughes, both of Greencastle, Ind., July 9, 1932, at Greencastle, Ind.

PERSONALS

Dr. B. T. Buckley (Iowa '32) has located at Bode, Iowa.

Dr. L. H. Wright (Corn. '14) is now associated with E. R. Squibb & Co.

Dr. L. E. Andres (O. S. U. '32) has selected Tipton, Ind., for a location.

Dr. W. L. Tanner (O. S. U. '32) has entered general practice at Van Wert, Ohio.

Dr. Ralph Case (Wash. '32) has located in Prescott, Ariz., for general practice.

Dr. H. E. Gerdes (San. Fran. '14) has been appointed City Meat Inspector of Phoenix, Ariz.

Dr. Herbert M. Tabbut (Iowa '32) will be located at Pelican Rapids, Minn., during the summer months.

Dr. E. H. Gloss (Iowa '31) has entered partnership with Dr. L. E. Stock (Iowa '29), at Alexandria, Minn.

Dr. C. Kelnhofer (West. '05) has resumed practice at Medford, Wis. He was located in Eau Claire for a time.

Dr. Frank R. Booth (Mich. '32) has located on East Jackson Blvd., Elkhart, Ind., just east of the city limits.

Dr. Cecil P. Hodson (Iowa '32) has entered partnership with Dr. H. A. Smith (McK. '09), of Anderson, Ind.

Dr. E. E. Patterson (Gr. Rap. '01), of Detroit, Mich., is recovering from a very severe attack of herpes zoster.

Dr. Albert Long (Harv. '95), formerly of Brooklyn, N. Y., is now at 9 Overlook Road, Melrose Highlands, Mass.

Dr. Chas. C. Gluhm (O. S. U. '31), formerly of Lexington, Ky., is now at 625 Prytania Ave., Hamilton, Ohio.

Dr. A. T. Gilyard (Corn. '07), of Waterbury, Conn., attended the 25th reunion of his class, at Ithaca, N. Y., June 17-18.

Dr. J. R. Karr (O. S. U. '32) has opened an office at Coshocton, Ohio. He spent two weeks at Camp Knox, Ky., in June.

Dr. A. M. Casper (Chi. '93), B. A. I. inspector-in-charge at Cedar Rapids, Iowa, was retired from active service July 1.

Dr. M. W. Emmel (Iowa '19), of the Alabama Polytechnic Institute, is attending summer school at Michigan State College.

Dr. W. E. Jennings (Corn. '31), of Kansas State College, was a recent visitor at Cornell University, while on a leave of absence.

Dr. R. C. Calkins (Chi. '08), of Rock Island, Ill., officiated as judge at the eighth annual Rock Island Pet Show, held June 30.

Dr. Olof Norling-Christensen (O. S. U. '32) has entered small-animal practice in Chicago. He is located at 1904 West North Avenue.

Dr. Seth D. Johnson (Corn. '29), of New York State Veterinary College, has recuperated from a recent operation for appendicitis.

Dr. M. Shipley (T. H. '17), of Phoenix, Ariz., recently accepted a position as sanitary officer in the Health Department of his city.

Dr. C. E. DeCamp (Corn. '24) has accepted a position as Director of the Veterinary Division of Rare Chemicals, Inc., at Napera Park, N. Y.

Dr. A. Buckmaster (Gr. Rap. '08) has resumed practice at Montpelier, Ind., after having been with the Geo. H. Lee Co. for over two years.

Dr. T. A. Newlin (K. S. C. '28), formerly of Perth Amboy, N. J., has opened an office in Dover, N. J., and will engage in general practice there.

Dr. Geo. W. Hare (Gr. Rap. '98), of Allegan, Mich., recently captured an albino mole. The animal, pure white in color, was taken in a spear trap.

Dr. A. B. Musser (O. S. U. '09), of Wapakoneta, Ohio, injured his spine when he fell down the basement stairway at the Hotel Koneta on June 29.

Dr. A. A. Rohrer (O. S. U. '32) has taken over the practice of Dr. F. M. Sharp (O. S. U. '11) at Worthington, Ohio. Address: 4768 North High Street.

Dr. W. T. Oglesby (Iowa '31) has accepted a position in the Department of Physiology and Pharmacology, Michigan State College, East Lansing, Mich.

Lt. J. J. Regan (Corn. '15) is Director of Quality Control for the Dairymen's League Coöperative Association, Inc., with headquarters in New York City.

Dr. John E. Hickey (Chi. '13), of Juneau, Wis., has announced his candidacy for the office of Register of Deeds on the Democratic ticket at the September primary.

Dr. F. M. Norris (Ont. '09), formerly of Reedley, Calif., has been transferred to Elk Grove, Calif., on meat inspection for the State Department of Agriculture.

Dr. Lloyd D. Jones (Iowa '31), of Hampshire, Ill., has accepted an appointment in the U. S. Bureau of Animal Industry, and has been assigned to meat inspection.

Dr. W. N. Armstrong (Ont. '94), of Concord, Mich., was elected president of the Jackson County (Mich.) Retail Druggists Association, recently organized at Jackson, Mich.

Dr. W. F. Straker (Ind. '15), of Greenville, Ohio, has been appointed Municipal Meat Inspector, under the provisions of a regulation recently adopted by the Greenville Board of Health.

Dr. Lester R. Barto (U. P. '30), who has been at the University of Pennsylvania Veterinary Hospital the past year, is now at the Dr. J. B. Engle Veterinary Hospital, Summit, N. J.

Dr. W. T. Brinker (O. S. U. '30), of Columbiana, Ohio, has accepted a position as veterinary inspector, in the Division of Dairy and Meat Inspection, Health Department, Cleveland, Ohio.

Dr. H. J. Schmidt (Amer. V. C. '86), of New Bremen, Ohio, has been seriously ill with an infection of the left leg resulting from a kick received from an equine patient two months ago.

Dr. A. S. Alexander (Chi. '97), for 29 years a member of the faculty of the University of Wisconsin, was made Emeritus Professor of Veterinary Science by the Board of Regents in June.

Dr. S. T. Walton (O. S. U. '22), of the Henry Ford Hospital staff, Detroit, Mich., served as president of the Detroit Local Branch of the Society of American Bacteriologists during the past year.

Dr. T. A. Sigler (Ind. '02), of Greencastle, Ind., was guest speaker at a recent meeting of the Greencastle Rotary Club. His address covered recent developments in the field of veterinary medicine.

Dr. Robert S. MacKellar (N. Y. C. V. S. '94), of New York, N. Y., represented the American Veterinary Medical Association at the inauguration of Dr. William A. Boylan as the first president of Brooklyn College, Brooklyn, N. Y., on June 21.

Dr. R. Learmonth (Mich. '25) received the degree Master of Science from the University of Colorado last month. His major was done in the Department of Bacteriology and Public Health, and his minor in the Department of Clinical Pathology.

Dr. J. P. Iverson (San. Fran. '06), in charge of meat and food inspection and sanitation for the Health Department of Oakland, Calif., recently served as Acting Health Officer, during the absence of Dr. Hieronymus, head of the Department.

Dr. C. T. Guilfoyle (U. P. '11) has taken over the practice of Dr. Jay C. Hicks (Iowa '20), at Tucson, Ariz., while the latter is taking a vacation to rest up for his political campaign this fall. Dr. Hicks is a candidate for the office of County Supervisor.

Dr. C. M. Prentice (O. S. U. '12), of Clyde, Ohio, captured a porcupine while on a fishing trip near Saint Ignace, Mich., with his son, the last week in June. The animal is to be stuffed and mounted and then placed in the museum of the local library at Clyde.

Dr. H. E. Moskey (U. P. '20), of the U. S. Food and Drug Administration, addressed the Maryland State Poultry Association, at the University of Maryland, on June 9, 1932. The subject was "The Control of Misbranded Poultry Remedies." Approximately a thousand members were in attendance.